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HEALTH ASPECTS OF PESTICIDES ABSTRACT BULLETIN is a monthly publication of the Technical Services Division of the Environmental Protection Agency.

The purpose of this publication is to foster current awareness of the major worldwide literature pertaining to the effects of pesticides on humans. The publication represents a monthly review of more than 1150 domestic and foreign journals. Subjects and authors of abstracted articles are cross-referenced in an annual index issue. Reprint requests should be directed to the author of the complete article.

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73-2027. Loftas, T. (Author address not given). **The oceans have become the sinks of the world.** *Ceres 5(1): 35-39; 1972.*

Bio-accumulation, one of the major problems of pollution in the ocean, has been shown to occur with DDT and other organochlorines. Associated with this problem is the fact that stress can force the release of stored chemicals in various organisms, and previously unknown harmful effects can be exerted. A number of fish kills have demonstrated the fact that these chemicals are not diluted as theoretically predicted, but remain concentrated in certain areas. Unprecedented international cooperation will be required to ban the ocean dumping and other practices which presently threaten the survival of this last important resource.

73-2028. Gerlow, A. R. (U. S. Dept of Agr., Washington, DC). **The economic impact of cancelling the use of 2,4,5-T in rice production.** *Economic Research Service, ERS-510, U.S. Government Printing Off., 1973, pp. 1-11.*

A $4.5 million reduction in farm income would be expected in Arkansas, Louisiana, and Mississippi deltas if the use of 2,4,5-T to control weeds in rice were cancelled. Effective herbicides that are not harmful to rice or nearby cotton and soybean crops have been difficult to find and experience with 2,4,5-T has led to its successful use. For every dollar spent on 2,4,5-T, a return of approximately $11 is expected. Since there are no milling facilities in Mississippi, high quality rice, produced with the aid of 2,4,5-T, is necessary to attract outside buyers. In addition disruption of existing domestic marketing practices and adverse affects on U.S. dollar markets abroad could result from limits on the use of 2,4,5-T. Major rice-producing areas in Texas, California, and southwestern Louisiana have been able to use less expensive herbicides effectively on indigenous weeds and would not be adversely affected by a cancellation of 2,4,5-T.

73-2029. Tahori, A. S. (Ed.) (Israel Inst. for Biol. Res., Ness-Ziona, Israel). **Fate of Pesticides in Environment.** *Gordon and Breach Science Publishers, New York-London-Paris, 1972, 572 pp.*

Fate of Pesticides in Environment is volume six in the series *Pesticide Chemistry* representing the proceedings of the Second International Congress on Pesticide Chemistry of the International Union for Pure and Applied Chemistry (IUPAC), held at Tel Aviv, Israel, February 22-26, 1971. The congress was attended by over 700 scientists representing 35 countries. The current volume contains papers and recommendations from workshop sessions on: factors influencing the fate of pesticides in soils; chemistry and metabolism of terminal residues of organochlorine, organophosphorus, and carbamate compounds, as well as fungicides; and the use of radioactive tracers for the study of fate of pesticide residues in food chains. Also included are papers from symposia on interpretation of basic chemical and toxicological data involved in development of pesticide residue tolerances and biochemistry of pesticides in relation to mammalian toxicology and pharmacology. Relevant papers have been included in this issue of *Health Aspects of Pesticides.*

73-2030. Van Tiel, N. (Plant Protection Serv., Wageningen, The Netherlands). **Organochloride insecticides: some observations on environmental and regulatory aspects.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 237-244.* (4 references)

DDT and cyclodiene compounds cause concern over pesticide side effects. Three categories of side effects can be defined: the development of resistance (basically caused by a change in population within the same species from the susceptible to the resistant strain and resulting from selection pressure of the pesticide); the interaction between plant and animal life and its abiotic environment, as long as this interaction occurs in the presence of pesticide residues at biologically active levels; and the presence of residues in food and subsequent intake of same by man. This final side effect necessitates the establishment and enforcement of tolerance levels and use patterns. The coverage of terminal residues under current legislation is difficult to assess. All legislation should allow for the addition of various metabolites as they become identified. Organochlorines often cause unintentional residues in animal products as a result of feeding animals fodder crops or feed treated with pesticides. However, organochlorines are often replaced with less persistent alternatives which are usually less stable and whose metabolism is not as well understood. Banning organochlorines must not be done without thoroughly studying the need for the pesticide in a given country and the side effects of substitutes.

73-2031. Korte, F. (Inst. Oekol. Chem., Schloss Birlinghoven, Germany). **Recommendations of workshop on chemistry and metabolism of terminal residues of organochlorine compounds.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 293-294.*

The following recommendations were proposed. The terms chlorinated hydrocarbons or organochlorine pesticides should be replaced with the chemical name for each compound. The ambiguous term of persistence should be replaced with the concept of function time rhythm. A break-down product present in minute quantities should not be placed under regulatory procedures unless specific strong reasons exist for so doing. While much is known about some of these pesticides, very little is known about others; this must be considered when setting tolerance levels. Critical reviews are needed on specific compounds. Further research is needed in the areas of: organochlorine pesticide breakdown in plants, the troposphere, and the stratosphere;

organochlorine compound residues in animal fat, milk, butter, and cheese; the relation of DDT to the environment with an eye toward establishing an acceptable benefit-risk equation for man and the environment; control of global chemical dispersions; the mode of pesticide actions, including synergistic and antagonistic effects; the enzyme induction by chlorinated pesticides.

73-2032. Robinson, J. R. (Canada Agr. Res. Inst., London, Ontario, Canada). **The choice of radioisotopes for studying the fate of pesticides.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 389-403.* (13 references)

The purpose of a proposed experiment plays a part in selecting the radiotracer technique to be used. Experiments concerning the mode-of-action of the pesticide on the biochemistry of the target organism, the chemical fate of the pesticide within the target, or the ultimate fate of the pesticide in unintended targets in the ecosystem dictate the selection of different radiotracer techniques. Before selecting the ideal radioactive label for the experiment, the advisability of labeling one of the elements already in the pesticide or of introducing by addition or substitution some element not originally present must be evaluated. In choosing a suitable radioisotope the availability and acceptable cost are factors easily decided. However, the properties of the radioisotope require consideration. These include physical properties such as radio half-life, type of radiation emitter, and energy requirements. Chemical properties include chemical half-life, biological half-life, presence of volatile forms, and residues. The ease with which the isotope can be handled may also determine selection.

73-2033. Lu, F. C. (Food Additives Unit, WHO, Geneva, Switzerland). **The role of WHO in conjunction with FAO in the development of pesticide residue tolerances.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 433-442.* (12 references)

Since 1966 FAO and WHO committees have met to consider various pesticides which leave residues in food. Acceptable daily intake (ADI) values, i.e., the maximum amount that can be ingested daily over a lifetime in food without appreciable risk to the consumer, have been determined. Tolerance levels for residues in food have been recommended. There are specific levels for the various stages of food production, including harvesting, storage, transport, marketing, food preparation, and consumption. Total diet studies, conducted to determine whether the consumer is at risk, have revealed that thus far the amount of pesticide reaching the consumer is considerably below ADI values. The Codex Alimentarius Commission has been established to protect consumer health and ensure fair practices in the food trade. One of its sub-committees is charged with proposing international tolerance levels. A second group establishes priorities among pesticide problems to be considered. The IUPAC has been helpful in providing necessary information for developing satisfactory analytical methods and supplying knowledge of the chemical nature of terminal pesticide residues used for establishing tolerance levels.

73-2034. Winteringham, F. P. W. (Joint FAO IAEA Div., Vienna, Austria). **Some biochemical research needs implied by the joint FAO/WHO programme on pesticide residues.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 479-494.* (22 references)

The growing need for food has increased the agricultural use of chemicals. Human pesticide exposure generally results from ingestion of food containing trace residues of the pesticide or its metabolites. Joint FAO/WHO meetings study the nature and magnitude of pesticide residues occurring after correct usage and establish whether these levels of contamination are tolerable. Identification of all significant products of pesticide degradation and metabolism is difficult. Even DDT has many unidentified derivatives. Organophosphorus insecticides degrade into a wide range of derivatives likely to be found by their multiple attack in vivo. Carbaryl is one of the few carbamate insecticides for which a somewhat complete account of the fate of known doses in animals can be given. The practice of applying herbicides and plant growth regulators to relatively mature plants necessitates studying their effects in food products. For long term effects the proportion of pesticide stored in lipophilic or bound form in adipose tissues must be determined. Research is necessary using normal agricultural pesticide concentrations where special conditions of local dietary and agricultural practices are considered, where the normal route of exposure is used in testing, and where the chronic effects of some metabolites can be studied in isolation. In total diet studies the total terminal residue will generally be larger than that determined analytically and, while limiting factors usually confine the study to single compounds, dietary residues consist of traces of more than one pesticide and their various derivatives.

73-2035. Anonymous. **Environmental pollution and the veterinarian.** *J. S. Afr. Vet. Med. Ass. 42: 277-279; 1971.*

Since the veterinarian studies and tests environmental contaminants, detects indicators of possible hazards of these contaminants to man, and gives practical advice to the public and health authorities on the use of pesticides, he should be well informed and also be represented, as he is in S. Africa, in the legislative body governing pesticide use. When deleterious effects have been definitely proven, as in the cases of dieldrin and methyl-mercury compounds, restrictions should be placed on their use and more acceptable compounds should be substituted. More work is necessary to determine the value of controversial pesticides like DDT.

73-2036. Shizuoka Prefectural Government (Shizuoka, Japan). **[Pesticide pollution.]** *Kogai Hakusho (The White Paper on Public Nuisance), 1972, pp. 156-167.* (Japanese)

A total sum of 4,782 million yen (approximately $16,737,000) was spent on 342 kinds of agricultural chemicals in Shizuoka Prefecture between October 1970 and September 1971. The per family use of pesticide in 1970 was 36,400 yen ($127.49) and was 4.8% of the total farm management budget. Between 1967 and 1971 the main chemical in pesticides was organic mercury. Organophosphate chemicals replaced mercury after it was banned except for seed disinfection. After 1971, carbamate insecticides replaced the legally banned BHC and DDT, and Saturn (benthiocarb) and MO (CNP) replaced PCP. Aldrin and endrin were replaced by diazinon. The prefectural policies for administration of agricultural chemicals, residue standards, the monitoring system of pesticide use, guidance, general inspection of cooperative facilities, promotion of safety movements and precautionary measures, enforcement of preventive measures, and systematic food residue inspections by analysis equipment are briefly discussed. Plans for inspection and control of chemical residues in the groundwater and rivers of Shizuoka Prefecture are discussed.

73-2037. Moore, E. E. (Pesticides Program, Div. of Environ. Serv., State Dept. of Health, Frankfort, KY 40601). **Pesticides sales and usage in Kentucky—1968.** *Pestic. Monit. J. 6(4): 379-387; 1973.* (6 references)

In Kentucky during 1968, 135 pesticide compounds were applied for agricultural and nonagricultural use in a volume of approximately 3.9 million pounds technical material (excluding most pesticides formulated for home, lawn, and garden use). This amounted to about 0.5% of the Nation's total consumption of pesticides. The pesticide poundage used in Kentucky, of which 67% was herbicides, was applied to an estimated 1 million acres, 4% of the State's land. This included over 900,000 lb of herbicides applied to rights-of-way throughout the State by various utility companies. Fourteen of the 135 base compounds sold constituted 73.3% of the total poundage used. These included, in order of volume, methyl bromide, maleic hydrazide, atrazine, DDT, 2,4-D, chlordane, sulfur, copper sulfate, aldrin, sodium chlorate, TDE, carbaryl, malathion, and methoxychlor. (Author abstract by permission)

73-2038. Anonymous. **Public health pesticides.** *Pest Contr. 41(4): 17-50; 1973.* (9 references)

In an annual release of the Center for Disease Control information is updated on compounds which are effective and can be used safely in the control of disease vectors and rodents. The use of DDT has been banned in the U.S. except for limited agricultural situations or when health officials feel that DDT is needed to control a disease vector. Dieldrin, chlordane, and aldrin are presently being examined by the Environmental Protection Agency. Excellent control of mosquitoes has been achieved in trials with ground-applied space treatments of pyrethrins (12%) synergized at 1 to 5 with piperonyl butoxide. Increased resistance to organophosphate compounds in mosquito larvae has forced many agencies to depend on adulticides. Houseflies have generally become resistant to both organochlorine and organophosphate compounds. Improving environmental sanitation has become the primary means of control. Resistance and susceptibility of particular species in particular areas also determine the choice of compounds for fleas, ticks, bedbugs, lice, and cockroaches. A number of organophosphates and organochlorines can be used to control venomous arthropods. Rodenticides must be considered supplemental to and not a substitute for good management in the control of rats.

73-2039. Anonymous. **Pesticides in perspective.** *Pesticides 6(12): 33-34; 1972.* (4 references)

Increased use of pesticides and fertilizers will be necessary to achieve the massive expansion needed in food production according to a recent study by the UN Food and Agriculture Organization (FAO). Disruption of the agricultural industry, which is at present sustaining 52% of the world's population, by denying the use of pesticides could imperil the living standards of tens of millions of people. Crop losses are at present at a level as high as 40% in some countries, and the large-scale use of pesticides is one of the main factors in preventing much greater losses. The use of pesticides has also made possible the control of certain insect-borne diseases. The majority of established pesticides have no adverse effect on man, animals, or the environment when used only in amounts sufficient to kill target pests although excessive amounts can be harmful. All pesticides should be applied carefully and in accordance with the manufacturer's instructions.

73-2040. Anonymous. **Pesticides and the environment.** *Pesticides 6(12): 35-38; 1972.* (12 references)

Most of the concern over the levels of pesticides in water has been generated by hasty reports of fish kills, of which only 2.5% have been attributed to pesticides in the U.S. since 1960, and limited laboratory studies such as the one in which the photosynthesis of four marine phytoplankton was inhibited by levels of DDT 1000 times greater than those found in the ocean. Although it is a well-known fact that residues of pesticides enter water, a study by the U.S. Geological Survey indicated that no pesticide concentrations in excess of the limits permissible in public water supplies were present in streams in western U.S. Varying soil characteristics and the wide range of available pesticides make evaluation of the effects of these chemicals on soil difficult. Most often cited contamination of soil by persistent organo-

chlorines is not necessarily an environmental problem since these chemicals generally do not move to ground water or streams except through transport on soil particles. Increasing yields on farms which extensively use herbicides indicates that soil fertility has not been damaged through the use of these pesticides. With the exception of four species of birds, wildlife has not been detrimentally affected by the use of pesticides.

73-2041. Anonymous. **Pesticides, clean food and human health.** *Pesticides 10(12): 41-43; 1972.* (10 references)

The possible risks associated with pesticide use in terms of human health are those of acute poisoning and chronic toxicity. In developed nations a very small percentage of farm accidents and of all poisonings is due to pesticide exposure. Few pesticides can be so poisonous that acute symptoms are observed, and these are safe if used properly. Long-term dietary studies are carried out for each pesticide to determine potential chronic toxicity, and from these residue tolerances are established which incorporate large safety factors. Market-basket sampling in several countries has indicated that levels of organochlorines and other pesticides are well below the acceptable daily intakes. Although DDT is known to concentrate in human fat, workers exposed to this chemical have not experienced any ill effects on health. The quality and quantity of food have reached a level never before observed as a result of the use of pesticides. The assumption that naturally grown foods are better than those produced by conventional methods overlooks the toxins and diseases carried on foods which have been eliminated through the use of pesticides. These chemicals have also dramatically reduced insect-borne diseases.

73-2042. Anonymous. **The development of a new pesticide.** *Pesticides 6(12): 44-48; 1972.* (1 reference)

An intensive research and development program, costing an average of $5 million and extending over a period of about 6 years, is required to bring a compound to the stage of a marketable pesticide. The synthesis of about 10,000 chemicals and initial screening of many of these is required for each successful entry into the pesticide market, and patent coverage is being used up during the development process. Once a compound has shown apparently useful activity, and relatively low toxicity to animals, birds, and fish has been established, field trials are run to evaluate it in situations representing actual use conditions. Acute, subacute, and chronic toxicity studies are conducted with a number of species and toxicity to microorganisms in soil determined. While these are being carried out, the cost of manufacturing the chemical by a satisfactory process is estimated. Residue studies in crops and various segments of the environment are carried out, and eventually tolerances are established.

73-2043. Anonymous. **Organophosphorus insecticides.** *Pesticides 6(12): 49-50; 1972.* (6 references)

The organophosphates have a narrower range of activity against insects, a shorter duration of activity, and a more rapid breakdown in plants and animals than other large classes of insecticides. A single dose of the earlier representatives is more toxic than that of most organochlorines, but compounds have been developed that have a low order of mammalian toxicity. Investigations are in progress to find organophosphates which can be used in integrated control methods since selective activity is a characteristic of some of these compounds. The control of pests on fruit, rice, and cotton has relied on organophosphates, and since many countries depend on one of these crops for their economic prosperity, the benefits are obvious. A special feature of some of these insecticides is the property of being absorbed by the plant and transported within it. This allows surface material to be washed off and only those insects which attack the plant to be affected.

73-2044. Anonymous. **Organochlorine insecticides.** *Pesticides 6(12): 51-54; 1972.* (11 references)

Organochlorine insecticides and acaricides differ considerably in chemical structure and properties and are particularly suited for overcoming a wide variety of pest problems. Although organochlorines are generally considered persistent chemicals, degradation does take place in the soil, and repeated applications do not result in build-ups of residues. In most cases organochlorines are not transported to aerial portions of plants even if root uptake is observed. Levels in foods do not exceed the acceptable daily intakes at present, and the current levels of those compounds found in humans do not appear to be harmful. Adverse effects on beneficial insects have been observed, and temporary adverse effects in aquatic life have been reported from gross contamination of waters by accident or careless use of organochlorines. Effects on wildlife have freqently been exaggerated. Beneficial effects of these pesticides are the well known increase in agricultural output and drastic reductions in insect-borne diseases.

73-2045. Anonymous. **Herbicides.** *Pesticides 6(12): 55-56; 1972.* (8 references)

Herbicides have been extremely useful in achieving fully mechanized farming. Selectivity can be a characteristic of the chemical, as found in phenoxy acids which affect broad-leaved but not grassy plants, or achieved by placement. Foliage-applied herbicides act on actively growing plants, while soil herbicides act on germination and have a longer effect duration. Great economic benefits have been derived from the use of herbicides, particularly in highly developed nations where labor is scarce. Since herbicides are generally of low mammalian toxicity and are applied early in the life of the crop or only on weeds, health hazards are minimal. A number of

residue studies have indicated no build-up of various herbicides in soil.

73-2046. Gupta, D. S.; Kaushik, S. N.; Kadyan, A. S. (Haryana Agr. Univ., Hissar, India). **Chemical pest control needs overhauling.** *Pesticides 6(12): 74-76; 1972.*

Since a great deal of development is required before pest control methods other than application of chemicals will be effective against most pest invasions, pesticides must continue to be the prime tool. Overhauling and remodeling the chemical methods of pest control are preferable to eliminating them. Toxicity and residue hazards, as well as detrimental effects to beneficial insects, can be eliminated by judicious use of the compounds available. Development of new broad spectrum insecticides which do not affect beneficial insects would reduce the problem of compatibility which arises from the need to use two or more insecticides.

73-2047. Chadha, D. S. (Central Committee Food Standards, India). **Pesticide residues in foods—their needs, present status, and principles for laying down tolerance.** *Pesticide 6(12): 83-87, 96; 1972.*

Pesticide residues may be controlled for the protection of the consumer by various general and legal means. Tolerance limits, established from the toxicity data and average food intakes and considering the levels found on crops, are used by many countries and have been established for grains in India. Although animals are used in most of the toxicological studies, data on exposed workers is helpful in establishing safe limits. Tolerances are legal limits and are generally much lower than safe limits and somewhat higher than levels expected to occur. Analytical procedures capable of detecting and identifying any kind of residue in food will have to be developed in order to monitor pesticide residues properly. The development of selective pesticide treatments which do not interfere with beneficial insects and mites will have to be substituted for the continued increases in pesticide applications resulting from resistance.

73-2048. Deshmukh, S. N.; Chawla, R. P.; Dutta, S. C. (Pestic. Residue Lab., Punjab Agr. Univ., Ludhiana, India). **Balance sheet of DDT.** *Pesticides 7(3): 24-26; 1973.*

DDT has been useful to mankind for three decades and saved millions of lives by providing more food and eliminating vectors of disease. Although its persistence presents environmental problems, this characteristic has made it a very attractive insecticide. Foodstuffs from many countries have contained DDT, but levels are not considered injurious and resulting residues in humans appear to be on the decline. Drinking water has likewise not contained levels of DDT known to be hazardous. The fear that accumulation of the pesticide in the ocean may adversely affect oxygen production is based on experiments in which extremely unrealistic levels were used. Substitution of newer insecticides for DDT will undoubtedly bring higher costs to farmers and public health agencies. Until an ideal substitute is found, DDT should continue to be employed with prudence in situations where it is judged to have a high benefit to risk ratio.

73-2049. Harvey, G. R. (Dept. Chem., Woods Hole Oceanogr. Inst., Woods Hole, MA 02543). **Source of PCB's.** *Science 180(4091): 1122; 1973.* (1 reference)

Worldwide observations of polychlorinated biphenyls (PCBs) in the environment do not support the published hypothesis that DDT is an unrecognized source of PCB, according to a Letter-to-the-Editor. All PCBs found in the environment are the 54 to 60% chlorinated mixture; di- and trichlorobiphenyls which could be from DDT are almost never found. Since the ratio of PCBs to DDT in the atmosphere, water, and biota of the East Coast and the Atlantic is greater than 10, DDT would have to be converted in high yield to only penta- and hexachlorobiphenyls, impossible by vapor-phase photolysis.

73-2050. Rabson, R.; Plimmer, J. R. (Biol. Branch, Div. Biomed. Environ. Res., U. S. Atomic Energy Comm., Washington, DC 20545). **Photoalteration of pesticides: summary of workshop.** *Science 180(4091): 1204-1205; 1973.*

A workshop was held at the National Academy of Science, Washington, D.C., April 6-8, 1972, by the U.S. Atomic Energy Commission and the U.S.D.A. to evaluate the importance of photochemical alterations in pesticides and to determine the possibility of using such information for the improvement of future strategies of pesticide use. In general, photochemical degradation products are less toxic to mammals and insects than the parent compound, but there are notable exceptions like dieldrin. Few of the reactions are documented, and residue monitoring systems do not detect their products. Volatility, type of application, soil characteristics, and other factors influence the exposure of pesticides to light. Photochemical oxidation, including the reaction of free radicals with oxygen to give a peroxy radical, is an important aspect of the phenomenon. Photoreduction, photodimerization, halogen bond cleavage, and enhancement of phenol acidity may also be significant in the natural environment. Research in the area of photochemical alteration appears to be decreasing although much more must be known before standardized estimation and prediction of photoalterations can be formulated.

73-2051. Ishikura, H. (Min. of Agr. and Forestry, Agr., Forestry, and Fish. Res. Council, Japan). **[Re-examination of technologies for utilizing agricultural chemicals and the themes in the future, a case study of technology assessment.]** *Shokubutsu Boeki (Plant Protect.) 27(1): 15-21; 1973.* (Japanese)

The Science and Technology Agency of the Japanese Government decided in 1971 to assess technology of agricultural chemicals, among other things. Similar studies had been conducted in the U.S. and the United Kingdom. Agricultural chemicals were selected because of wide popularity and number of problems which need to be solved. The assessment was carried out by eight specialists in agricultural chemicals. The group re-examined the technologies of agricultural chemicals and studied the impact of agrichemicals. Evaluations of negative aspects led to suggestions on how to control detrimental effects. The suggestions are gradually being instituted by the government.

73-2052. Anonymous. **[Amendment of a part of Standard of Safe Use of Agricultural Chemicals and its official noufication.]** *Shokubutsu Boeki (Plant Protect.) 27(1): 37-39; 1973.* (Japanese)

The amendment to the Standards (tolerances) of Agricultural Chemicals in/on Foods Act enlarged the coverage of agricultural chemicals from 12 to 18 (fenthion, fenitrothion, chlorobenzilate, dicofol, tricyclohexyltin hydroxide, and inorganic bromide newly included) and the foods from 25 to 29 kinds (sweet potato, broad bean, wheat, and corn). The Standards of the Safe Use of Agricultural Chemicals Act was also amended to create new residue standards in/on crops. The newly established standards are: 0.2 ppm of BHC, 0.2 ppm DDT and 0.3 ppm parathion on sweetpotato, wheat, broad bean, and corn; 0.1 ppm of EPN on turnip (leaf and root) and burdock; 0.5 ppm of malathion on apple, summer orange (peel and edible part), Japanese pear, Japanese radish (leaf and root), and burdock; 2.0 ppm of malathion on Chinese cabbage; 0.1 ppm of diazinon on cucumber, cabbage, spinach, peach, pumpkin, and sweet potato; 1.0 ppm of carbaryl on cabbage, summer orange, Japanese pear, peach, Japanese radish, and sweet potato; 2.0 ppm of dicofol on cucumber and summer orange; 3.0 ppm of dicofol on grape, apple, strawberry, Japanese pear, and Satsuma orange; 2.0 ppm of chlorobenzilate on grape, apple, strawberry, summer orange, Japanese pear, peach, and Satsuma orange; 0.2 ppm of fenitrothion on cucumber, tomato, grape, apple, strawberry, tea (green), Japanese pear, peach, rice, pumpkin, green, pepper and Satsuma orange; 0.05 ppm of fenthion on potato and rice; 5.0 ppm of inorganic bromine and 2.0 ppm of tricyclohexyltin hydroxide on apple and Japanese pear. Dieldrin, aldrin, and endrin must not be detected on wheat, broad bean, and corn.

73-2053. Hawkes, H. A. (Appl. Hydrobiol. Sect., Univ. Aston, Birmingham, England). **Disposal by dilution?—An ecologist's viewpoint.** *Soc. Appl. Bacteriol. Symp. Ser. 1: 149-179; 1971.* (100 references)

Although dilution can solve some waste effluent problems, it is not suitable for the disposal of nonbiodegradable or toxic substances like pesticides. Permissible levels for pesticides have been established on the basis of LC50 values, but these do not reflect adverse effects resulting from chronic exposure to lower levels of the chemicals. Pesticides differ widely in their susceptibility to degradation, but many are quite persistent. Although accumulation in river water is not a common phenomenon, bottom sediments may contain 100 times the concentration of pesticides in the overlying water. Aquatic organisms are capable of taking up many of the compounds and storing them in tissue, and in this way they are concentrated at higher and higher levels via the food chain. Besides the obvious dangers involved in the concentration of toxic materials in the food of man, the death of various organisms can significantly alter the ecology of a waterway.

73-2054. Garrison, A. W.; Case, F. N.*; Smiley, D. E.; Kau, D. L. (Oak Ridge Nat. Lab., Oak Ridge, TN). **The effect of high pressure radiolysis on textile wastes, including dyes and dieldrin.** *Advan. Water Pollut. Res., Proc. Int. Conf. 5th, 1971, pp. II-37/1–II-37/10.* (16 references)

High pressure radiolytic oxidation (8×10^6 R/hr at 1500 to 2000 psi) was capable of destroying 98% of the dieldrin residue expected to occur in wool processing effluents and was also able to reduce color, BOD, COD, and pH of textile wastes. Irradiation alone removed 64.5% of the dieldrin, and oxygen or air pressure removed only 30 to 32%. With actual wool processing wastes, 95% of the dieldrin was removed by irradiation alone and 99% was removed upon addition of oxygen. It appeared that the minimum time required for removal was less than 10 minutes.

73-2055. Wilson, D. J.; Locker, D. J.; Ritzen, C. A.; Watson, J. T.; Schaffner, W.* (George Hunter Lab., Vanderbilt Univ. Hosp., Nashville, TN 37232). **DDT concentrations in human milk.** *Amer. J. Dis. Child 125(6): 814-817; 1973.* (19 references)

Samples of human milk obtained from white, middle-class donors from Long Island, Rochester, Chicago, Lexington, Nashville, Memphis, and Los Angeles contained an average of 0.17 ppm total DDT. Only four samples had no detectable residues, and a high level of 0.83 ppm was recorded. Long Island donors had residues significantly lower than those in other cities. A lower amount of DDT was found in the milk of women who employed exterminators, rather than applying pesticides themselves, but no significant difference was observed in milk residues from those who had and had not used pesticides in their homes and gardens. Those who used butter rather than margarine had significantly lower residues, and seasonal variation in DDT levels was noted. The concentration of total DDT was much higher in milk taken from an empty breast, than in milk taken before the breast was emptied. Although no evidence of harmful effects has been recorded for DDT in infants, the fact that the residues in human milk are higher than those recommended for cow's milk necessitates further study.

73-2056. Szokolay, A.; Uhnak, J. (Vyskumny ustav hygieny, Bratislava, Czechoslovakia). **Expozicia cloveka reziduam DDT a HCH z mliecneho tuku na Slovensku a navrh na ich tolerancie. [Exposure of man to DDT and BHC residues from milk fat in Slovakia and proposal of acceptable daily intake levels.]** *Bratisl. Lek. Listy 58(5): 558-565; 1972.* (16 references) (Slovak)

One hundred seventeen butter samples taken over a period of 17 months from dairies in six Slovak towns were analyzed for DDT and BHC content. From the figures obtained, human exposure was calculated as 0.189 mg BHC and 0.118 g DDT/kg body weight/day based on the average consumption figures for milk and butter fat per inhabitant of Slovakia. These figures are comparable with the respective levels found in other European countries, except that DDT intake generally exceeds lindane intake. The intake of chlorinated hydrocarbons from all sources in Slovakia is probably similar to that in the U.S. (0.0014 mg/kg from all foods except beverages). Although lindane has a higher acute toxicity, DDT persists longer, which is why they have almost the same acceptable daily intake levels. New two-stage norms are proposed for maximum acceptable daily intake values of DDT and BHC residues in milk and milk products based on strict toxicological criteria. Two stages are required because currently the soil is still too contaminated from unrestricted pesticide application in the past years and the strict final norms could not be met immediately. The DDT norms for milk (2.5% fat), for butter (85% fat), and for milk products (100% fat) call for 0.027, 0.926, and 1.00 mg/kg DDT up to Dec. 31, 1974, and 0.015, 0.528, and 0.570 mg/kg thereafter. The norms for lindane call for 0.029, 0.994, and 1.00 mg/kg up to Dec. 31, 1974, and for 0.021, 0.726, and 0.730 mg/kg thereafter.

73-2057. Saschenbrecker, P. W. (Animal Pathol. Div., Canada Dept. Agr., Guelph Area Lab., Ontario, Canada). **Levels of DDT and PCB compounds in North Atlantic fin-back whales.** *Can. J. Comp. Med. 37(2): 203-206; 1973.* (20 references)

DDT and its metabolites and polychlorinated biphenyls were found in blubber tissue samples of 12 North American fin-back whales caught by commercial establishments at various points off the Canadian Atlantic coast. Residues detected in ppm included: DDT (0.351-1.302); TDE (0.007-0.506); DDT derived compounds (0.198-0.454); polychlorinated biphenyls (0.012-0.0185). Dieldrin was not found in any sample. Since the proportion of blubber is about 30% of the total body weight, one whale may store 38 grams of DDT compound and 2.7 grams of polychlorinated biphenyls. Gas chromatographic results were confirmed by chemical derivatization.

73-2058. Duseja, D. R. (Utah State Univ., Salt Lake City, UT). **Adsorption-desorption and movement of picloram (4-amino-3,5,6-trichloropicolinic acid) in soils.** *Diss. Abstr. Int. 33(9): 4071B; 1973.*

Adsorption and desorption of picloram in soils were studied using both batch technique and soil columns. In batch studies the soils adsorbed 10.8 to 58.2% of the added picloram from 0.05-10.0 ppm aqueous picloram solutions. Adsorption was highly correlated with organic matter and sesquioxides content of the soils, and significantly correlated with cation exchange capacity. Equilibration time was 4-120 hr. An increase in the temperature from 17.7°C to 25°C caused increased adsorption; raising the temperature further

decreased adsorption. Adsorption followed the Freundlich model. Adsorption showed a close negative correlation with pH changes. From 41.0 to 71.8% of the initially adsorbed picloram could be eluted by two successive extractions with deionized water. Picloram in aqueous solution was not precipitated by addition of electrolytes. In soil divalent inorganic cations increased adsorption more effectively than monovalent cations. Studies on the effect of calcium chloride concentrations on picloram adsorption suggested an exchange type reaction with picloram acting as a cation. Column studies corroborated the findings of batch studies; inorganic salts did impede picloram movement in soils, presumably by increasing picloram adsorption. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-5622.)

73-2059. Brooks, T. M. (Memphis State Univ., Memphis, TN). **A study of the metabolism, degradation, and volatilization of endrin after application to soils.** *Diss. Abstr. Int. 33(1): 4160B; 1973.*

To determine the fate of endrin applied to soils thirty-seven compounds representing possible metabolic or degradation products of endrin were purchased or synthesized and their gas-chromatographic relative retention times (RRT) determined on three columns of different polarity. Field soil samples known to have been treated with endrin were collected, extracted, and analyzed; the RRT values obtained for the principal extracted components were compared with the values for the standards on the three columns. Thirteen of the standards matched with the RRTs of major components; 9 did not give GLC responses on the columns used; and 15 definitely were not among the major components of the soil extracts. During a study of volatilization of endrin and its metabolite, ERP1, from soils under controlled conditions, increases in temperature and percent moisture greatly increased volatilization. Increases in rate of air flow and addition of UV radiation only slightly increased endrin volatilization. ERP1 showed no detectable volatilization after 4 days. More endrin volatilized from the surface of a watch glass than from the surface of the soil, although the areas of the two surfaces were similar. Prolonged storage of endrin and related standards resulted in conversion of 5 compounds, but none of the conversion products matched with the RRT of a major soil extract component. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms,Order No. 73-6517.)

73-2060. Bode, L. E. (Univ. of Missouri, Columbia, MO). **Environmental factors influencing trifluralin diffusion in Mexico silt loam soil.** *Diss. Abstr. Int. 33(9): 4251B; 1973.*

An understanding of herbicide movement in soil is necessary for developing placement specifications for herbicides. Diffusion of radiolabeled trifluralin through Mexico silt loam soil was measured in diffusion cells under laboratory conditions. Preliminary studies indicated that trifluralin diffusion coefficients are independent of diffusion time and chemical concentration in the soil. Vapor diffusion contributed the major portion of total diffusion at bulk densities less than 1.2 g/cm^3. The magnitudes of vapor and solution diffusion are similar at bulk densities between 1.2 and 1.4g/cm^3. There is an exponential relationship between temperature and the diffusion coefficient in the temperature range from 4.4 to 49°C. Diffusion was low in air-dry soil for all temperatures, passing through a maximum at 8-15% moisture. When the air-filled porosity was reduced below approximately 50%, diffusion declined rapidly. A prediction model employing 15 terms and which accurately described the response surface of trifluralin diffusion coefficients was developed. Diffusion coefficients of 3.8×10^{-11} to 2.8×10^{-6} cm^2/sec were found. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-7015.)

73-2061. Abernathy, J. R. (University of Illinois, Urbana-Champaign, IL). **Linuron, chlorbromuron, nitrofen, and fluorodifen adsorption and movement in twelve selected Illinois soils.** *Diss. Abstr. Int. 33(10): 4616B; 1973.*

Adsorption and movement of linuron, chlorbromuron, fluorodifen, and nitrofen were studied on twelve Illinois soils under greenhouse, growth chamber, and field conditions, using adsorption, pH, electrical migration, desorption, temperature, and soil-TLC characteristics. Soil isotherm data indicated about 1.5 times as much chlorbromuron as linuron was adsorbed by each soil studied. Adsorption was correlated with organic matter content and cation exchange capacity of soils. Slightly more nitrofen than fluorodifen was adsorbed on all soils. Herbicides were adsorbed readily from low-pH solutions by Drummer soil and carboxymethylcellulose (cation exchanger), whereas diethyl-aminoethylcellulose (anion exchanger) more readily adsorbed herbicides from pH 12 solution. All herbicides migrated toward the cathode in low pH solutions and toward the anode in high pH solutions. More linuron and chlorbromuron than fluorodifen and nitrofen were desorbed from Drummer soil. Linuron and chlorbromuron adsorption was enhanced at 60°F on Plainfield and Drummer soils while linuron and chlorbromuron adsorption was greater at the higher temperature on the Pittwood soil. Nitrofen and fluorodifen adsorption was greater at the higher temperature for all three soils. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-9861.)

73-2062. Waller, J. B. (Iowa State Univ., Ames, IA). **Interaction of three Iowa soils with the organophos-**

phorus insecticide phorate and its metabolites, and the effect of these compounds on Southern corn rootworm larvae. *Diss. Abstr. Int. 33(10): 4848B; 1973.*

Five grams of soil (Clarion, Webster, or Harps) were incubated with 0.5 ml of $10^{-4}M$ phorate, which approximates the current usage rate (2 lb/acre). Both unlabeled and ethoxy-l-^{14}C-labeled phorate were used in these experiments. Incubations were carried out at 10, 20, and 30°C for periods up to 16 days. The higher the temperature the more rapid the loss of phorate. After 2 days at 30°C only 15% of the added phorate remained, approximately 30% was adsorbed to the soil, 35% oxidized to the sulfoxide, and 20% to the sulfone. Soxhlet extraction of soil incubated with ^{14}C-labeled phorate for 2 days resulted in a 70% recovery of the added activity, again leaving 30% unextractable and presumably adsorbed to the soil. Phorate sulfoxide and sulfone were the only metabolites of phorate in soil extracts detected by gas-liquid chromatography and autoradiography of thin-layer chromatography plates. The oxygen analog pathway does not appear to be operative in this soil system since phoratoxon and its sulfoxide and sulfone were never detected by any of the procedures used. Hydrolysis constitutes a minor pathway of phorate breakdown. Less than 2% of the added ^{14}C-labeled phorate was recovered in the aqueous phase from a chloroform:water (2:1 v/v) extract of soil incubated for 48 hr. Soil sterilization studies showed that oxidation of phorate to its sulfoxide was mainly nonbiological, whereas the sulfoxide to sulfone conversion was mainly biological. Phorate was the least toxic compound, and phoratoxon the most toxic of the 5 oxidative metabolites to the southern corn rootworm. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-9497.)

73-2063. Khalilov, I. M. (Tadzhik Inst. Agr., USSR). **Peredvizheniye i posledeystviye gerbitsidov. [Effects and aftereffects of herbicides.]** *Dokl. Akad. Nauk Tadzh. SSR 16(2):56-59; 1973.* (Russian)

In-depth migration of fluometuron in the soil was studied by using cucumbers as indicator plants, and aftereffects of fluometuron, nitrofor, planavin (nitralin), and herban (norea) were investigated on barley. The cucumber indicator test revealed little or no effect of 113 mm of precipitation on the migration of fluometuron applied to the soil surface at a rate of 3.2 kg/ha. About 50% of the total quantity applied was retained in the upper 7 cm soil layer after a total precipitation of 240 mm. Fluometuron and nitrofor, applied at the respective rates of 2.5-2.75 kg/ha and 4 kg/ha, partially inhibited barley for five months and stimulated barley growth afterwards. Herban and planavin had no residual effect and stimulated growth. The herbicides investigated are completely inactivated in the soil during the first year after their application.

73-2064. Mulla, M. S.; Norland, R. L.; Westlake, W. E.; Dell, B.; St. Amant, J. (Dept. Entomol., Univ. California, Riverside, CA 92502). **Aquatic midge larvicides, their efficacy and residues in water, soil, and fish in a warm-water lake.** *Environ. Entomol. 2(1): 58-65; 1973.* (4 references)

During an efficacy study of several chemical larvicides, residues of chlorpyrifos were monitored in bottom mud and in bottom-dwelling and frequenting fish. After treatment of one-half of a lake with the compound at 0.2 lb/surface acre, mean residues in the water reached 0.6 ppb within 24 hr and residues in the top 1 inch of mud averaged 0.3 ppm. No residues were observed in deeper mud and the residues in the top section fell to < 0.1 ppm in 28 days. Movement of the toxicant into untreated areas was evidenced by a decline in midge populations. Residues in largemouth bass and bluegill fluctuated between 0.1 and 0.2 ppm during the two weeks after treatment while a mean of 0.8 ppm was found in bottom feeding channel catfish within 2-3 weeks. Similar water and mud residues were obtained when the entire lake was treated at the same rate.

73-2065. Miller, R. W.; Faust, S. D. (Dept. Soil Sci. Biometeorol., Utah State Univ., Logan, UT 84321). **Sorption from aqueous solution by organo-clay: III. The effect of pH on sorption of various phenols.** *Environ. Lett. 4(3): 211-223; 1973.* (13 references)

Results of a study of several phenolic compounds and two phenoxy acetic acids on Bentone 24 and 18 C (bentonite clay coated with dimethylbenzyl octadecylammonium chloride or a dodecylamine) suggested that sorption of these 2,4-D type compounds is pH dependent and probably associated with the extent of protolysis of the phenolic group. Relative sorptions were similar on both coated clays, but the sorption on 18C was only slightly more than 50% as much as on Bentone 24. Maximum sorption occurred frequently where protolysis was less than 50%.

73-2066. Lichtenstein, E. P. (Dept. Entomol., Univ. Wisconsin, Madison, WI 53706). **Persistence and fate of pesticides in soils, water and crops: significance to humans.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 1-22.* (30 references)

No absolute half-life can be assigned to pesticides in the environment since their existence depends on the chemical nature of the pesticide, the type of soil, temperature, light, moisture content, microorganisms, and numerous other environmental factors. The water solubility of the pesticide and the absorptivity of the soil type determine to what extent the chemical moves through the soil. Aldrin, parathion, and other commonly used insecticides adhere to loam soil particles, and only trace amounts can be removed with water. However, they can be transported on particles of soil. Low water solubility pesticides are being degraded microbiologically

within lake or river bottom mud while some detergents prolong the life of insecticides in soils. Polychlorinated biphenyls seemingly prevent detoxification of certain insecticides, thus synergizing their activity, and interfere with analyses of certain chlorinated insecticides. Soils rich in organic matter allow little absorption of insecticides by plants. Although the penetration of some insecticides into plant roots may be large, the translocation of the chemicals to green plant parts is minimal. Aldrin and DDT at 125 ppm disrupted DNA, RNA, and protein synthesis in tissue cultures of human cells; aspirin and sodium chloride gave similar results. At 10 ppm these disruptions had mostly disappeared. It is suggested that research be directed toward determining the pharmacological significance of pesticidal pollutants in biological systems and the potential interaction of synthetic chemicals on subcellular levels.

73-2067. White, J. L.; Cruz, M. (Dept. Agron., Purdue Univ., Lafayette, IN). **The role of soil colloids in the behavior and fate of pesticides in soils.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 23-45.* (37 references)

Pesticide-soil interaction is greatly influenced by the colloidal properties of soil particles. Various spectroscopic studies of surface reaction mechanisms between organic molecules and aluminosilicate minerals suggest mechanisms involved in adsorption, degradation, and detoxification of pesticides by soil components. Infrared studies suggest that pesticide molecules and water molecules competing for adsorption sites may explain losses by desorption and volatilization. In the acid environment of clay surfaces certain pesticides such as the chlorotriazines are subject to reactions that the organic cations of amitrole are not. The chlorotriazines are hydrolyzed to produce the protonated hydroxytriazine. In soils rich in montmorillonite or vermiculite this protonated hydroxytriazine is strongly held and the amount of *s*-triazine and its degradation product in the soil water will be reduced. The methoxytriazine (prometone) and methylthiotriazine (prometryne) are more readily protonated upon adsorption on montmorillonite than the chlorotriazine (propazine). Failure to acknowledge this protonated hydroxytriazine state makes extraction procedures to remove triazines and their degradation products from the soil very difficult.

73-2068. Plimmer, J. R. (Plant Sci. Res. Div., ARS, USDA, Beltsville, MD 20705). **Photochemistry of pesticides: a discussion of the influence of some environmental factors.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 47-76.* (29 references)

Intermolecular reactions, determined largely by environmental conditions surrounding the pesticide molecule, to a large extent direct the decomposition of pesticides on the ground surface. Solvent or surrounding molecules may participate in these reactions. A change in the energy required to trigger photodecomposition may be caused by adsorption of the pesticide molecule to the surface of silica or clay mineral, thus causing a change in the maximum molecular absorption wavelength. Spectral changes resulting from adsorption to silica have been measured in DDT derived compounds, 2,4,5-T, *o*-chlorobenzoic acid, 2,3,6-trichlorobenzoic acid, *o*-chlorobenzonitrile, dichlobenil, dioxins, propham, chloropropham, 3,4-dichloroaniline, propanil, trifluralin, 4-chlorophenol, 3,4,5-trichlorophenol, 2,3,5,6-tetrachlorophenol, and PCP. Photosensitized decomposition occurs when a sensitizer molecule absorbs light energy and transfers it to a reacting species. In photosensitized oxidation singlet oxygen may be produced by interaction of a sensitizer with oxygen. Singlet oxygen can cause reactions at sites remote from those at which it is generated. Therefore, photochemical reaction is possible some distance away from the absorbing surface.

73-2069. Wheatley, G. A.; Suett, D. L.; Hardman, J. A. (Nat. Vegetable Res. Sta., Wellesbourne, Warwick, England). **Some factors influencing the persistence of chlorfenvinphos in soil.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 77-85.* (24 references)

An area of sandy loam soil was rotary-cultivated, and the insecticide, Birland Granules, containing 9.2% chlorfenvinphos, was spread to give 1.85 kg a.i. ha^{-1}. The insecticide was worked into the soil to depths of 0-4 cm or 0-10 cm. Carrots and radishes were sown. Interval soil samples were taken until after the harvesting of the crop. Factors affecting the persistence of chlorfenvinphos were studied. Samples stored for seven days at room temperature averaged a 28% loss of chlorfenvinphos residue. Hexane extracts of treated soil similarly stored suffered no change in chlorfenvinphos concentrations. Results indicate that leaching would not move any significant amount of the insecticide below the maximum depth of cultivation within a season. There was no measurable effect of depth of incorporation of chlorfenvinphos on its subsequent rate of decay. Chlorfenvinphos is relatively persistent in the highly organic fen soils, and care is needed to avoid accumulation of residues under abnormally dry conditions in mineral or fen soils or if crops like carrots are treated too frequently on the same land. This insecticide persists in the soil without change while soil temperatures are below 6-8°C and must be considered when applying the insecticide the following season. The absence of any marked deceleration or acceleration of loss rate in the autumn or spring, respectively, associated with the winter stability, suggests that the primary breakdown of chlorfenvinphos in this soil was biologically dependent.

73-2070. Saltzman, S.; Yaron, B. (Dept. Soils and Water, Volcani Inst. Agr. Res., Bet Dagan, Israel). **Para-**

thion adsorption from aqueous solutions as influenced by soil components. *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 87-100.* (9 references)

Soil adsorption of parathion from aqueous solution was studied using C-14 labeled parathion. Adsorption isotherms were obtained for 20 soils, Na-kaolinite, Na-montmorillonite, organic material, and glass beads. The organic material and Na-montmorillonite showed strong affinity for parathion (1-9μg/ml), and were far from approaching saturation. At the highest solution concentration the Na-kaolinite was close to saturation; the glass beads were saturated. It is concluded that the main soil components affecting parathion adsorption are the expanding-lattice clays and the organic matter, which present linear adsorption isotherms. Parathion adsorption on non-expanding lattice clays and on non-colloidal soil materials is reduced and may be expressed by an adsorptive isotherm fitting the Freundlich equation. The λ-value (distribution coefficient), and k-value (derived from the Freundlich isotherm) are constants which characterize the capacity of the soil for parathion.

73-2071. Horowitz, M. (Div. Weed Res., Volcani Inst. Agr. Res., Newe Ya'ar, P.O. Haifa, Israel). **Ecology of herbicides under irrigated subtropical conditions.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 101-115.* (74 references)

Bioactivity and degradation of herbicides in warm irrigated soil are reviewed. The type of organic matter and pH influence bioactivity in soils low in organic matter. In subtropical climates photodecomposition and volatilization are important dissipation factors. Disappearance is inversely related to soil adsorption and directly related to soil moisture. If sprinkler irrigation immediately follows herbicidal spraying, the loss is minimized. The water solubility of the herbicide and the amount of irrigation water directly affect the depth of leaching. Conversely, soil adsorption inversely affects the depth of leaching of herbicides. Movement upwards and laterally and diffusion are factors in the disappearance of herbicide residues. Most herbicides undergo microbiological degradation, and conditions favorable to microbiological activity aid in this process. Non-biological degradation has been observed in atrazine, trifluralin, and benefin. Since microbiological activity decreases with increasing soil depth, the greater the depth of the herbicide in the ground, the slower the degradation.

73-2072. Montgomery, M. L.; Klein, D.; Goulding, R.; Freed, V. H. (Oregon State Univ., Corvallis, OR 97331). **Biological degradation of pesticide wastes.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 117-125.* (10 references)

Biological soil degradation has been studied as a possible means of detoxifying pesticide wastes. Site selection must meet these criteria: there must be a minimum risk of contaminated water leaving the testing area; microorganisms capable of degrading the waste material to be detoxified must be in the soil; the land used must be of low economic value. The waste concentrate used for this study resulted from the manufacture of 2,4-D and thus contained unreacted 2,4-dichlorophenol, unrecovered 2,4-D, and a considerable amount of sodium hydroxide. The waste material was diluted and spread at the rate of 300 pounds per acre of 2,4-D and 900 pounds per acre of dichlorophenol. Significant reductions of the chemicals occurred during the first few months; these were greatest in the surface two inches of soil. About 80% of the applied chemical was gone the first year. Microorganism counts shrank initially but returned to normal levels in about 4-6 months. Proper management, consisting of maintenance of moisture and temperature, accelerated the rate of degradation.

73-2073. Upchurch, R. P. (Monsanto Co., St. Louis, MO 63166). **Perspectives on herbicide behavior in soil.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 127-138.*

The major reasons for studying herbicidal behavior in soil are: to know that the herbicide is safe for man and the environment, to learn how soil properties affect rates or methods of application, and to discover new pesticides with superior performance. Behavior patterns and data gathering methods are reviewed. The following factors can be studied under growth chamber conditions: the influence of soil on phytoactivity and general herbicidal behavior, the influence of clay minerals (soil texture) on phytotoxicity, the persistence of a given herbicide, the impact of soil pH, and the relative leachability of herbicides. It is hard to characterize the activity of herbicides in vapor form. The lag phase concept in relation to microbial detoxication has not been used extensively of late. In placing herbicides the following points must be considered: weed control effectiveness, crop tolerance, reliability of performance, different interactions with plant roots and plant shoots, photodecomposition and volatilization losses at the soil surface, and the periodicity of herbicidal breakdown with season changes. Soil organic matter binds with the herbicide, preventing losses by leaching, volatilization, or chemical and microbial attack, at the same time protecting the plants from the herbicide. The amount of herbicide used can affect its degradation. Most laboratory studies use higher rates than would be normal for a typical use situation, thus providing no concrete facts, but rather only a basis for hypothesizing what happens under normal conditions.

73-2074. Johnsen, R. E.; Lin, C. S.; Collyard, K. J. (Dept. Entomol., Colorado State Univ., Fort Collins, CO 80521). **Influence of soil amendments on the metabolism of DDT in soil.** *IN: Fate of Pesticides in Environ-*

ment, Gordon and Breach, London, 1972, pp. 139-156. (19 references)

Manure was added to soil in an effort to study any effect it might have on DDT metabolism due to stimulation of indigenous soil microbial populations. A Colorado loam soil with 14% clay, pH 7.9, and 1.9% organic matter was used either alone or with added cattle manure. With no manure added, DDT metabolized slowly, and recovery decreased with time. With even a low rate of manure added, the DDT disappeared rapidly. A considerable amount of DDE was produced when no manure was added, but no DDE was recovered when manure was used. This may be due to the manure acting as an energy source resulting in anaerobic conditions and subsequent reduction of DDT to TDE and not DDE, which will not form anaerobically. To determine whether cattle manure contributed microorganisms to the soil mixture, the effects of using autoclaved and non-autoclaved manure were compared. In all cases recovery was decreased with autoclaved manure. Mercuric chloride added to soil treated with DDT and manure inhibited the conversion of DDT to TDE or other metabolites. The use of glucose solution in place of manure as the energy source resulted in essentially no breakdown of DDT. Under conditions where DDT was converted to TDE, the TDE apparently metabolized further since DDMA, DDMS, and DBP were found. However, when TDE was added to soil and incubated with cattle manure, TDE was recovered unchanged. DDE behaved similarly.

73-2075. Sieper, H. (E. Merck, Darmstadt, Germany). **Residue analysis and degradation of morphactins.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 157-174.* (10 references)

Various Flurenol (flurecol) compounds have been studied by colorimetric methods. After the active ingredient was extracted from the sample, cleaned up by preparative chromatography on silica gel, and transformed to a blue derivative of fluorenone, it was measured at 597 nm. Flurenols degrade rapidly in cereals, grass, water, and soil, as evidenced by the degradation curves (half-life= 1-2 days). The definitive degradation product, 2-chlorofluorenone, was found in the soil after chloroflurenol methylester application. Irradiation (uv) in an aqueous or benzene solution also results in 2-chlorofluorenone formation.

73-2076. Kaufman, D. D. (Plant Sci. Res. Div., ARS, USDA, Beltsville, MD 20705). **Degradation of pesticide combinations.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 175-204.* (60 references)

Increased or decreased persistence and the formation of complex residues sometimes result from the use of various pesticides in simultaneous or successive applications. Whether or not these effects are desirable depends upon the ultimate result of the interaction involved. The formation of persistent residue complexes is undesirable, and decreased persistence of pesticide residues is needed. Certain methylcarbamate and organophosphate insecticides inhibit the enzymatic hydrolysis of several phenylcarbamates, acylanilides, and acetamide type herbicides by soil microorganisms, thus increasing their persistence in soil. Controlled persistence of readily degradable pesticides may facilitate better and longer pest control with reduced applications and application rates, and ultimately reduce accumulation of undesirable residues. Careful selection of pesticide combinations or formulations can aid in the control of residual toxicity. In deliberate use of pesticide combinations factors such as reliability and effectiveness, as well as the economics of their production, their toxicology, and the ecological acceptability of their individual and their combined residues must also be considered.

73-2077. Korte, F. (Org. Chem. Inst., Bonn Univ., Bonn, Germany). **Chemistry and metabolism of terminal residues of organochlorine compounds—state of the art.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 205-222.*

Pesticides undergo chemical change due to chemical, photochemical, and enzymatic reactions. A substance is considered unduly persistent whenever a measurable quantity of it continues to exist in some discernible chemical form. Photoisomerization or photodechlorination are the only known abiotic reactions of organochlorine compounds in the environment. Concerning pesticide metabolism, very little is known of the metabolites; many are observed but not identified. Not much is known about the degradation of metabolites to the persistent end products, either under atmospheric conditions or in higher plants. On the contrary, much is known about the metabolism of some pesticides like DDT in a number of organisms. Water soluble metabolites of aldrin and dieldrin, though frequently mentioned in the literature, are largely unidentified, apart from the phenolic metabolites of lindane and DDA. It is noted that pesticides have different metabolic behaviors in animals and plants. This difference appears in the metabolic end products formed in the different species and in the metabolic rate. Therefore, the chemical structure of a compound is not the deciding factor in determining the fate of the compound under environmental conditions.

73-2078. Brooks, G. T. (Agr. Res. Council, Unit Invertebrate Chem. Physiol., Univ. Sussex, Brighton, England). **Degradation of organochlorine insecticides; problems and possibilities.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 223-236.* (26 references)

A study of the chemical and biological transformations of organochlorine compounds is made extremely

difficult due to the small amounts of available metabolic products. Biotransformation products may be produced by in vivo experiments, use of tissue homogenates or partially purified enzyme systems, and use of chemical systems modeling enzyme reactions. The relevance of photochemical reactions to environmental changes in toxicant composition and the fact that photochemical reactions conducted in the presence of oxygen sometimes produce products similar to those formed by microsomal mixed function oxidase enzymes illustrate the usefulness of such reactions with organochlorine compounds. Due to the importance of anaerobic microbial biotransformations regarding the final disposal of orgachlorine compounds, reagents such as chromous chloride, which appear to mimic such processes, will be of increasing interest.

73-2079. Chopra, N. M. (North Carolina Agr. Tech. State Univ., Greensboro, NC 27411). **Breakdown of chlorinated hydrocarbon pesticides in tobacco smokes: a short review.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 245-261.* (27 references)

Degradation products of DDT in tobacco smoke have been studied in two phases: a study on the pyrolysis of DDT in a nitrogen atmosphere at 900°C, and a study on the breakdown of DDT in DDT treated tobacco smokes. Isolated products from the first phase included DDT, DDT derived compounds, TDE, DDM, *cis-* and *trans-p,p'*-dichlorostilbenes, chloro-*bis*-(*p*-chlorophenyl)methane, *o,p'*-dichlorotoluene, *p,p'*-dichlorobiphenyl, hexachloroethane, tetrachloroethylene, trichloroethylene, carbon tetrachloride, chloroform, and dichloromethane. Three reaction mechanisms explain formation of these compounds. The following pyrolysis products were obtained from DDT treated tobacco: DDT, DDT derived compounds, TDE, DDM, *cis-* and *trans-p,p'*-dichlorostilbenes, *bis*-(*p*-chlorophenyl)-methane, *p,p'*-dichlorobenzophenone, chlorobenzene, chloroform, and methyl chloride. Dichloromethane was not detected. The amount of chloroform in tobacco smoke was about 1.6% of the amount of DDT present in tobacco, but the amount of methyl chloride (2,000μg/g tobacco smoked) was independent of the amount of DDT in the tobacco. Results demonstrate that stronger bonds have better survival rates during smoking than do weaker bonds. The breakdown of DDT in tobacco smoke must be approached with regard for the significance of the amount of degradation product arising from the pesticide degradation versus the amount of degradation product which is a natural component of the tobacco smokes. Also to be considered is the extent to which non-toxic metabolic products of the pesticide give rise to toxic degradation products in tobacco smokes.

73-2080. Ware, G. W.; Estesen, B.; Cahill, W. P. (Dept. Entomol., Univ. Arizona, Tucson, AZ). **Effects of two years DDT moratorium on agricultural residues in Arizona.** *IN: Fate of Pesticides in Environmental, Gordon and Breach, London, 1972, pp. 287-292.*

The withdrawal of DDT from active use in Arizona agriculture caused a drop in the total DDT residue in green alfalfa from an average of 404 ppb in 1967 to 45 ppb in 1970. DDTR residues in the soil were 2.29 ppm in 1969 and 2.66 ppm in 1970, upholding the thought that DDT has a 10-12 year half-life. Total DDT residues in beef fat, which are an indirect indicator of DDT residues in animal products, which in turn are direct indicators of the agricultural use of DDT, dropped from an average of 0.97 ppm in 1968 to 0.49 ppm in 1970. It must be noted that total DDT residues in alfalfa, beef fat, and milk fat were only 35% DDT derived compounds in 1967, but generally exceeded 85% DDT derived compounds in 1970. The future problem from DDT residues will be thus due to DDT derived compounds rather than to DDT itself.

73-2081. Aharonson, N.; Resnick, C. (Ministry of Agr., Jerusalem, Israel). **The dissipation in plants of some new organophosphorus insecticides.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 333-345.* (8 references)

Dursban (chlorpyrifos), a nonsystemic insecticide, was compared to the more persistent nuvacron (monocrotophos) and phosvel (leptophos). Dursban degraded rapidly with a residue half life of 5-7 days on tomatoes. No degradation occurred for nuvacron or phosvel over a 28 day period after treatment, probably because of translocation of the residues within the plants. *Spodoptera littoralis* can be better controlled by the systemic organophosphorus insecticides, however, these are a more persistent class of pesticides with a slower degradation rate. The ability to analyze an organophosphate molecule to which a halogen atom is attached using electron capture and thermionic detectors allows detection and confirmation both to be undertaken on the gas chromatograph.

73-2082. Brown, J. R. (Univ. Toronto, Toronto, Canada). **DDT in the agricultural soil and human tissue in Norfolk County, Ontario.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 551-571.* (23 references)

DDT is used in Norfolk County to protect the tobacco crop. Blood samples were taken from the general population of the area, from farm workers, from persons living in an area where DDT was not widely used, and from workers at a pesticide formulation plant. DDT and its derivatives were slightly higher in blood concentration in persons living in the Norfolk County area, but not so high as the concentration in the blood of persons working at a pesticide formulation plant. Samples of soil were taken from land used for a variety of crops. Soil concentrations of DDT and its derivatives

were greatest in the top six inches of soil and declined rapidly at deeper levels. The slight accumulation of the pesticide and its metabolites was caused in part by the sandy nature of the soil. It is also suggested that dissemination occurred either by co-distillation with water from the soil or by removal from the surface as wind blown dust.

73-2083. Korolev, A. A. (Dept. Communal Hyg., I. M. Sechenov First Moscow Inst. Med., Moscow, USSR). **Ozonirovaniye kak metod obezvrezhivaniya vody, zagryaznennoy khimicheskimi soyedineniyami. [Ozonization as a method for the decontamination of water polluted by chemical compounds.]** *Gig. Sanit. 37(12): 78-83; 1972.* (51 references) (Russian)

Application of ozonization for the treatment of waters polluted with chemical compounds such as phenols, surfactants, other organic and inorganic matter, petroleum products, and pesticides is reviewed. Ozonization helped remove carbophos (malathion), parathion, ronnel, lindane, aldrin, and dieldrin. Quantitative destruction of 10 mg/l. concentration of carbophos by 26 mg of ozone per liter, and reduction of identical concentrations of parathion and ronnel to 0.7 and 0.1 mg/l. by 8-10 mg of ozone per liter were observed. It was also possible to decontaminate water containing 0.55 to 2.2 mg of lindane per liter by the use of ozone.

73-2084. Tsunoda, T. (Kushiro Fisheries Scientific Institution, Hokkaido, Japan). **[Aerial spray of herbicides–sodium chlorate residue.]** *Hokusuishi Geppo (J. Hokkaido Fish. Sci. Inst.) 29(2): 30-35; 1972.* (5 references) (Japanese)

Sodium chlorate ($NaClO_3$) herbicide granules containing 50% $NaClO_3$ and 50% sodium hydrogen carbonate and clay shaped into 1-2 mm spherical or cylindrical particles were sprayed by helicopter in July 1971 in the basins of the Tadarui River. A total of 48,036 kg of herbicide was sprayed from 30 m above ground over 214.28 ha (224 kg/ha). Seven locations were investigated; water samples were taken immediately before and after spraying for up to 34 days, and residues were measured. The orthotoluidine method and spectrophotometry were used for analysis of $NaClO_3$. Some residue was detected at two locations, both downstream of rivers adjacent to the spray area. At one location 6.3 ppm was extracted one hour after spraying, 1.3 ppm four hours later, 0.4 one day later, and none after the second day. At another location 25.9 ppm was extracted one hr after spraying, 3.6 the next day, 1.8 two days later, 0.1 ppm on the 23rd and 27th days, and none after the 29th day. The concentration was not large enough to affect fish or other wildlife. No residue was extracted at any point 500 m or more away from the spray area.

73-2085. Schnitzer, M.; Khan, S. U. (Soil Res. Inst., Canada Dept. Agr., Ottawa, Ontario, Canada). **Reactions of humic substances with organic chemicals, N-containing compounds, and physiological properties of humic substances.** *IN: Humic Substances in the Environment, Marcel Dekker, Inc., New York, 1972, pp. 281-293.*

Adsorption of pesticides by humic substances is generally studied with the slurry technique, but can be determined by dialysis. Elevation of temperature increases adsorption which is optimum at higher temperatures and pH values approximating the pKa of the pesticide. A review of the literature shows that hydrogen bonding and ion exchange are the primary mechanisms of adsorption although much remains to be learned about the nature of humic substances. In addition to adsorption capability sodium humate can act as a solubilizing agent for compounds like DDT.

73-2086. Nishimoto, T.; Uyeta, M.; Taue, S.; Chikazawa, K. (Kochi Prefect. Health Res. Inst., Kochi, Japan). **[Organochlorine pesticide residues and PCB in breast milk.]** *Igaku No Ayumi (Progr. Med.) 82(9): 974-975; 1972.* (7 references) (Japanese)

Approximately 50 ml of breast milk was collected from each of 26 women in Kochi and Nangoku between December 1971 and February 1972. The women, between the ages of 22 and 35, were all healthy and had had no special contact with organochlorines or PCBs. α-, β-, γ-BHC, p,p'-DDE, p,p'-DDT, and dieldrin were detected, but almost no traces of δ-BHC, p,p'-DDD(TDE), and heptachlor epoxide were found. An average of 0.03 ppm PCB was found in all samples as determined with KC-500 gas chromatography. The BHC and DDT contents in milk coincided with previously reported findings of residues in human adipose tissues and showed a 40-50% decrease compared to the investigation of 1971, while dieldrin scarcely decreased.

73-2087. Vardell, H. H.; Gillenwater, H. B.; Whitten, M. E.; Cagle, A.; Eason, G.; Cail, R. S. (Agr. Res. Serv., U.S. Dept. of Agr., Savannah, GA 31403). **Dichlorvos degradation on stored wheat and resulting milling fractions.** *J. Econ. Entomol. 66(3): 761-763; 1973.* (4 references)

Dichlorvos sprayed on wheat at 15 ppm resulted in deposits ranging from 2.4 to 6.0 ppm immediately after treatment. Residues decreased to 0.5 ppm or less during six weeks of storage in 3.5-m^3 bins. Temperature of the grain was an important factor affecting residue-degradation rate. There was little or no loss of residues from wheat stored for three months at $-18^{\circ}C$. The residues on wheat stored at $27^{\circ}C$ for 1 week were about equal to that stored in bins at 18 to $12.7^{\circ}C$ for 5–6 weeks. Residues on the milling fractions were greater on the bran and shorts than on the flour. The treatment did not adversely affect patent flour or bread baked from the patent flour. (Author abstract by permission)

73-2088. Leuck, D. B.; Bowman, M. C. (Southern Grain Insects Res. Lab., Agr. Res. Serv., U. S. Dept. of Agr., Tifton, GA 31794). **Chlorphoxim residues: their persistence in coastal bermudagrass.** *J. Econ. Entomol. 66(3): 798-799; 1973.* (22 references)

Test plots in a pure stand of coastal bermudagrass were sprayed with analytical grade chlorphoxim and its *O*-analog, as well as 2 lb AI/gal EC chlorphoxim at the recommended rates of 8 and 16 oz/acre as well as the excessive rate of 32 oz AI/acre. The residues of chlorphoxim in grass treated with EC at the highest rate declined from 84 ppm (wet basis) to 0.03 ppm during the first 14 days of weathering. The *O*-analog was detected only in the 0- and 1-day samples. Chlorphoxim residues had declined to 0.13 ppm or less at all treatment rates by the 7th day post-treatment and were practically undetectable after three weeks. Residues of a metabolite with a GC retention time 0.75 times that of chlorphoxim were detected in all samples through the 7th day post-treatment. The metabolite contained phosphorus and sulfur in the same ratio as in chlorphoxim. The *p*-value in hexane-acetonitrile was 0.070. A compound of similar analytical behavior was prepared by UV irradiation of dry chlorphoxim residues, but has not yet been detected. Small quantities of two other unidentified metabolites were also present.

73-2089. Vardell, H. H.; Cagle, A.; Cooper, E. (Stored Product Insects Res. and Develop. Lab., Agr. Res. Serv., U. S. Dept. of Agr., Savannah, GA 31403). **Phosphine residues on soybeans fumigated with aluminum phosphide.** *J. Econ. Entomol. 66(3): 800-801; 1973.* (2 references)

Phosphine gas from aluminum phosphide tablets is used in fumigation of soybeans against the cereal leaf beetle. To determine whether this procedure leaves residues in the soybeans, fumigations were conducted in triplicate in controlled-temperature chambers at 7.2, 12.8, 18.3, and 23.9°C. Aluminum phosphide pellets were introduced into the bottom of each drum to correspond to a rate of 180, 150, or 120 pellets/1000 bu. After fumigation times of 2.5 days, the lids were removed, and air was drawn through the soybeans with a fan for 24 or 48 hours. The highest residue found on soybeans after fumigation was 0.04 ppm; this was apparently due to the collection of some unreacted aluminum phosphide with the sample. Phosphine residues in all other samples did not exceed 0.002 ppm. Residues did not vary consistently with depth in the barrel or with aeration time. A tolerance for phosphine on soybeans has not been established; however, the highest value found in this test would fall within the limit of the tolerance established for phosphine on other commodities such as corn, barley, oats, and rough rice.

73-2090. Krueger, H. R.; Mason, J. F. (Dept. of Entomol., Ohio Agr. Res. and Develop. Cent., Wooster, OH 44691). **Phorate and aldicarb: effects of systemic fungicides on their levels in soybeans.** *J. Econ. Entomol. 66(3): 815-816; 1973.* (4 references)

Soybean plants grown in pots under greenhouse conditions were treated at the time of unfolding of the second trifoliate leaf cluster with 50 ml of water containing 5 or 20 mg of triarimol, plantvax (DMODC), carboxin, Ethirimal, RH-124, chloroneb, thiabendazole, benomyl, thiophanate-methyl, or thiophanate. One week later the soil in each pot was drenched with 50 ml of water containing either 9 mg of phorate or 3 mg of aldicarb. Phorate and its three oxidative analogs and aldicarb and its toxic oxidation products were measured by GLC. Phorate residues ranged from 61 to 147 ppm on the tenth day after insecticide treatment and 60-190 ppm on the twentieth day, regardless of fungicide treatment. Aldicarb residues were 55-174 ppm. There appeared to be no significant differences in the uptake or degradation to nontoxic metabolites of either phorate or aldicarb when used in combination with the fungicides tested. Phytotoxicity was produced by some of the treatments, especially at the higher fungicide levels, and in some cases this elevated the phorate and aldicarb levels which were based on leaf weights.

73-2091. Kuwatuka, S. (Nagoya Univ., Dept. of Agriculture, Japan). **[Pesticides in the soil.]** *Kagaku Kogyo (Chem. Ind.) 23(11): 81-88; 1972.* (62 references) (Japanese)

General chemical pollution, movement, adsorption in the soil, and residue and degradation processes are reviewed. In Japan the duration of chemical residue in the soil is relatively short because of high humidity and warm temperature; organophosphate residues decompose rapidly and usually disappear within several months. The half life of parathion is six days, or a 67% decrease in 17 days; the half life of dimethoate is two and a half days; that of diazinon is 20-80 days or in some cases less than 17 days. The degradation period for most herbicides is one to two months and depends on soil condition, water content, temperature, and pH. Gamma-BHC (lindane) decomposes 30% in six weeks with a water content of 60-80%; in 100% water almost no residue is found in six weeks. Seventy-five percent of DDT remains for more than six months under an aerobic condition. The addition of 1% alfalfa or creation of an anaerobic condition reduces DDT to 1% in 12 weeks. The half-life of PCP in a wet rice paddy is 12 days to several months; in soil with extremely limited carbon content almost no portion is degraded in 50 days. If soil is disinfected completely, residues will not be degraded. Bacteria and soil and the chemical process of residue degradation are briefly reviewed.

73-2092. Gorelik, L. D.; Kushchinskaya, I. N.; Nemets, S. M. (All-Union Sci. Res. Inst. Canning and Vegetable Drying Industry, USSR). **Vliyaniye tekhnologicheskikh**

rezhimov na soderzhaniye ftalofosa v konservakh. [Effect of technological conditions on the content of phthalophos in canned food.] *Konserv. Ovoshchesush. Prom. 4: 8-10; 1973.* (Russian)

The effect of washing, blanching, and sterilization on phthalophos residues was studied in canned apples, plums, cabbage, pears, green peas, and red beet. Apples and plums were dipped into 0.4% aqueous solution of phthalophos for three minutes before washing. Washing with five parts of 0.5% solution of sodium hydroxide to one part of fruit reduced the residue content by 50-55%. Blanching at a temperature of 80°C and at a liquid to fruit ratio of 5:1 removed 55% and 81% of the initial phthalophos content from plums and apples, respectively. Blanching with acidified water was least effective, while blanching with 100°C steam for five minutes had an efficiency of 48-49%. The rate of phthalophos destruction by sterilization ranged from 60 to 100% in different products and from 37.5 to 38% in cabbage. No phthalophos residues were detected in canned apples and plums stored over 12 months at 20°C temperature.

73-2093. Neuhaus, J. W. G.; Brady, M. N.; Siyali, D. S.*; Wallis, E. (P.O. Box 162, Lidcombe, New South Wales 2141, Australia). **Mercury and organochlorine pesticides in fish.** *Med. J. Aust. 1(2): 107-110; 1973.* (8 references)

Over 90% of the canned and fresh fish samples from markets in Australia contained mercury in levels below the recommended limit of 0.5 ppm. Although mercury residues in fish from areas of high industrial pollution were higher than in those from the open, they were much lower than those reported from polluted areas overseas. Residues in canned fish from Australia or overseas were comparable. Mean organochlorine residues were 0.005 ppm heptachlor epoxide, 0.084 ppm total DDT, and 0.042 ppm dieldrin. Less than 14% of the samples contained BHC, lindane, or HCB. Shellfish samples contained an average of 0.135 ppm total DDT, 0.037 ppm heptachlor epoxide, and 0.064 ppm dieldrin. Organochlorine residues found in surveys in other countries were similar to those found in the present study.

73-2094. Siyali, D. S.; Simson, R. E. (Anal. Lab., N.S.W. Dept. Health, Joseph St., Lidcombe, New South Wales 2141, Australia). **Chlorinated hydrocarbon pesticides in human blood and fat.** *Med. J. Aust. 1(4): 212; 1973.* (2 references)

Very high dieldrin levels were observed in fat and blood of symptomatic rural pest control operators. The highest blood level observed was 424 ppb, and 28% had a level in excess of 75 ppb. Heptachlor epoxide, DDT, and HCB (hexachlorobenzene) were found in much lower concentrations. Blood levels of organochlorines were good indicators of body burden if they remained high for 48 hours after exposure. Blood levels greater than 50 ppb were considered indicative of excessive exposure. Medical practitioners should be aware of the value of these screening tests and forward samples by the fastest method possible to the Department of Health.

73-2095. Pooler, Jr., F. (Div. Meteorol., Environ. Protect. Agency, Raleigh, NC). **Atmospheric transport and dispersion of pesticides.** *Nat. Tech. Inform. Serv. Com-72-10454, 1972, 20 pp.* (6 references)

A summary of the effects governing local and widespread distribution of pesticides in the biosphere shows that the local dispersion is almost entirely due to atmospheric transport, and that the atmosphere is only a contributor in the complex scheme of dispersion on a wide scale. Droplet size, the height at which the material is released, and the wind speed and atmospheric turbulence govern the deposition of pesticide in a given area. Frontal uplifting and penetrative convection are two principal mechanisms which lift some of the surface air mass containing pesticide from contact with the surface, thus reducing the deposition process. Precipitation, which often follows both lifting processes, can also deposit the impurities. Global distribution of a pesticide is difficult to predict, but generally an air mass does not change latitude so that a band of distribution would be expected. Dry deposition is probably the most important means of removal of DDT, the pesticide of primary concern on a global scale. The mean residence time in the atmosphere is probably about a week or two.

73-2096. Kamata, T. (Sch. of Hyg., Fac. of Med., Hiroshima Univ., Hiroshima, Japan). **[Hygienic studies on pesticide residues. Part 3. Determination of organochlorine pesticide residues in human tissues and other samples.]** *Nippon Eiseigaku Zasshi (Jap. J. Hyg.) 27(5): 439-443; 1972.* (13 references) (Japanese)

Farm produce from Hiroshima, Shimane, and Okayama Prefectures, animal fats, cow and human milk, and human tissue samples were collected and the organochlorine pesticide contents determined by gas chromatography. BHC and DDT residues were widely distributed. Values from farm products ranged from 0.05-0.18 ppm of BHC and trace-0.95 ppm DDT. The γ-isomer of BHC was most prominent, followed by α- and β. *p,p′*-DDT predominated; *o,p′*-DDT remained moderately and *p,p′*-DDE remained very little. No dieldrin was determined. BHC and DDT residues in bovine fat ranged from 5.6-12.1 and 0.15-0.25 ppm; in swine fat from 0.21-0.39 and 0.10-2.3 ppm. In animal fat almost all BHC was β-isomer and DDT was *p,p′*-DDE. In human milk BHC ranged from 0.07-0.16 ppm with an exception of 0.31 ppm. DDT ranged from 0.09-0.18 with an exception of 0.41 ppm. In the present investigation rice straw used for feed in polluted districts contained 0.28-0.63 ppm of BHC while that in unpolluted districts contained only 0.02 ppm. There was little difference in DDT content between polluted (0.02-0.09) districts and unpolluted

(0.01 ppm) districts. In human tissues which had been dried at 80 C for five hours both pesticides were found most abundantly in liver and kidney. The extracted fat from human tissue contained 2.3-11.5 ppm of BHC and 1.2-6.7 ppm of DDT. Almost all BHC was β-isomer, and the larger part of DDT-fraction was *p,p'*-DDE followed by *p,p'*-DDT, *o,p'*-DDT being very small.

73-2097. Kanazawa, J. (Agr. Tech. Res. Inst., Japan). **[Food chain and concentration in the body.]** *Nogyo Oyobi Engei (Agr. Hort.) 48(1): 164-168; 1973.* (Japanese)

A review of the following topics is presented: the food chain in the animal world; organic synthetic chemicals and residue concentration in marine animals, water fowls, land animals; organochlorine residues in cattle; residue effects on humans and the time lag of residue concentration in the body. The time that residues take to go through lower animals to higher animals and peak is considerably long. According to studies at M.I.T., the peak in the DDT spray and the peak of DDT residue in fish are approximately 11 years apart. The use of BHC was totally prohibited in Japan starting in 1970. The β-BHC concentration in unpolished rice in 1970 was one eighth of the previous year's, in milk was one third of the previous year. However, β-BHC in mother's milk peaked in 1971 and is now gradually decreasing.

73-2098. Goto, S. (Pesticide Residue Research Inst., Japan). **[Pesticide and residue characteristics.]** *Nogyo Oyobi Engei (Agr. Hort.) 48(1): 179-184; 1973.* (Japanese)

The amount of pesticide residue and extent of degradation depend on many factors such as type of produce, method of application, timing, method of cultivation, and weather conditions. Physical degradation by evaporation, wind dispersion, and rain washout are greater in the order of powder, solution, and emulsion forms. Most pesticides are organic and soluble in oil, and the residues on the surface of plants quickly permeate into the surface layer of plants. Although pesticides generally remain in the skin or the surface layer, water soluble pesticides infiltrate into deeper layers and sometimes, as ethylthiometon, are oxidized and circulate in the plant. Mercury easily concentrates in the carbohydrate layer of rice, and DDT and dieldrin concentrate in the oil of soybeans and peanuts. Produce that grows relatively quickly, like tomatoes and cucumbers, dilutes the residue as it grows. The degradation pattern in such produce is often predictable by plotting logarithms. Since many pesticides are insoluble and remain in the top 20 cm of the soil, deep tilling can dilute the residue to a certain extent. The main degradation process in the soil is evaporation. Sandy earth helps evaporation. Watery soil (paddies) helps the proliferation of anaerobic bacteria, and pesticide residues decompose quickly. Various components of BHC are degraded approximately 90% in 50 days in rice paddies. Soil or rice paddy pollution by BHC is relatively limited in spite of the large quantity used.

73-2099. Calderbank, A. (Jealott's Hill Res. Station, Bracknell, Berkshire, England). **Environmental considerations in the development of diquat and paraquat as aquatic herbicides.** *Outlook Agr. 7(2): 51-54; 1972.* (20 references)

An intensive investigation of the environmental effects of diquat and paraquat as aquatic herbicides was carried out in the U.S., England, Canada, Australia, and elsewhere. The studies showed that the two compounds have many advantages. In all trials they disappeared rapidly to below 0.1 ppm within a few days and to a non-detectable level within 10 to 14 days. The loss is attributed to the absorption and concentration of the herbicide in aquatic plants. As the weeds decomposed the water remained free of residues, probably because adsorption to bottom mud took place. The lowest TLm observed for fish was 2.1 ppm, which exceeds the highest application rate of 1.5 ppm. No mortalities have been observed in the field with fathead minnows, channel catfish, sunfish, and brown trout, and no significant residues were found in edible portions of the fish. In feeding experiments farm animals which might come in contact with treated herbage were not adversely affected by levels up to 400 ppm in the food for one month, and no residues were detected in meat or milk. Activated carbon and clay were effective in removing diquat and paraquat from water, indicating that ordinary water treatment methods would eliminate any contamination from reservoirs. An indirect effect of herbicide application, the death of fish from consumption of dissolved oxygen by decaying weeds, can be avoided by treating only small areas at one time.

73-2100. Bindra, O. S. (Dept. Zool.-Entomol., Punjab Agr. Univ., Ludhiana, India). **Pesticidal pollution of water.** *Pesticides 6(12): 77-82; 1972.*

Extensive studies have been made in many countries on pesticide residues in different waters. Organochlorines have been found in minute amounts in rainwater in England. Sub-surface waters generally do not contain residues, although well water contamination has resulted from seepage from industrial wastes or intensive agricultural use. Intentional and unintentional application and indirect incorporation through municipal and industrial discharge, run-off, and rain contribute the bulk of pesticide residues in surface waters. Adsorption by activated carbon is reported to be the most effective treatment for removing pesticides from water for drinking, although no methods eliminate the low-level, long-term contamination. Irrigation water is often contaminated with pesticides, but only the herbicides have been found detrimental. In India very little is known of the contamination of waters, but fish kills

have prompted the installation of treatment plants in pesticide manufacturing facilities.

73-2101. Moore, III, S.; Bruce, W. N.; Kuhlman, D. E.; Randell, R. (Coop. Ext. Serv., Coll. of Agr., Univ. of Illinois, Urbana, IL 61801). **A study of the sources of insecticide residues in milk on dairy farms in Illinois–1971.** *Pestic. Monit. J. 6(4): 233-237; 1973.* (5 references)

In 1971, a study was conducted to determine the sources of chlorinated hydrocarbon insecticide contamination to dairy cows in order to help dairy farms avoid, or recover from, a residue problem in their milk supply. Twelve dairy farms were selected from 40 herds surveyed initially in February 1971; the 12 farms had dieldrin milk residues ranging from low to medium to high. Samples of milk were collected from the 12 farms in February, March, and September; in March, samples of feed, well water, and soil were also collected. Cropping history, insecticide usage history, and cattle management practices were obtained for each farm for the preceding 10-year period, 1961-70. The following conclusions may be drawn from this study: the chances of dieldrin residues in milk exceeding the Food and Drug Administration's current administrative guideline of 0.3 ppm (fat basis) are greatest on dairy farms having a history of aldrin soil treatment within the last 6 or 7 years; hay and oat straw supply significant amounts of dieldrin to dairy cattle; in addition, roasted soybeans could be an important source of dieldrin contamination in milk; corn silage, commercial feed concentrate, and well water are usually not important sources of dieldrin contamination to dairy cattle. (Author abstract by permission)

73-2102. Butler, P. A. (Ecolog. Monit. Branch, Tech. Serv. Div., Off. of Pestic. Programs, U.S. Environ. Protect. Agency, Gulf Breeze, FL 32561). **Organochlorine residues in estuarine mollusks, 1965-72–National Pesticide Monitoring Program.** *Pestic. Monit. J. 6(4): 238-362; 1973.* (17 references)

Analyses of the 8,095 samples for 15 persistent organochlorine compounds showed that DDT residues were ubiquitous; the maximum DDT residue detected was 5.39 ppm. Dieldrin was the second most commonly detected compound with a maximum residue of 0.23 ppm. Endrin, mirex, toxaphene, and polychlorinated biphenyls were found only occasionally. Results indicate a clearly defined trend towards decreased levels of DDT residues, beginning in 1969-70. At no time were residues observed of such a magnitude as to imply damage to mollusks; however, residues were large enough to pose a threat to other elements of the biota through the processes of recycling and magnification. (Author abstract by permission)

73-2103. Miles, J. R. W.; Harris, C. R. (Res. Inst., Canada Dept. of Agr., University Sub Post Office, London 72, Ontario, Canada). **Organochlorine insecticide residues in streams draining agricultural, urban-agricultural, and resort areas of Ontario, Canada–1971.** *Pestic. Monit. J. 6(4): 363-368; 1973.* (6 references)

Organochlorine insecticide residues in water systems draining agricultural, urban-agricultural, and resort areas of Ontario, Canada, were compared by analysis of water, bottom mud, and fish collected during the period from mid-April to mid-October 1971. Insecticides detected were *p,p'*-DDT, *o,p'*-DDT, *p,p'*-TDE, *o,p'*-TDE, *p,p'*-DDE, γ-chlordane, dieldrin, endrin, endosulfan, heptachlor, heptachlor epoxide, lindane, and aldrin. Insecticide concentrations in water from all three areas were less than the "Maximum Reasonable Stream Allowances" for growing fish that are safe for human consumption. The concentrations of total DDT in the water were combined with water flow data to calculate the weekly rate of transport of total DDT at each sampling time. The greatest transport of total DDT was by the Muskoka River which drains the Muskoka Lakes resort area where DDT was used until 1966 for control of biting flies; a peak of 11.8 lb total DDT per week was recorded in May, but this transport quickly lessened, resulting in a May to October average of 1.9 lb total DDT per week. Corresponding figures for the Thames River (urban-agricultural) were peak 2.5 lb and average 0.4 lb total DDT per week and for Big Creek (agricultural), peak 0.5 lb and average 0.2 lb per week. The ratio of concentration of total DDT in mud to total DDT in water was as great as 800; total DDT in fish to total DDT in water was as great as 1 million. The ratio of *p,p'*-TDE to *p,p'*-DDT was <1 in water but >1 in bottom mud, indicating possible dechlorination of *p,p'*-DDT to *p,p'*-TDE in the bottom mud. Polychlorinated biphenyls (PCBs) were present in the urban-agricultural area samples of bottom mud and fish at levels up to 217 ppm and about 0.4 ppm, respectively. (Author abstract by permission)

73-2104. Carey, A. E.; Wiersma, G. B.; Tai, H.; Mitchell, W. G. (Tech. Serv. Div., Office of Pestic. Programs, Environ. Protect. Agency, Beltsville, MD 20705). **Organochlorine pesticide residues in soils and crops of the corn belt region, United States–1970.** *Pestic. Monit. J. 6(4): 369-376; 1973.* (16 references)

In order to determine the levels of organochlorine pesticides in the Corn Belt region of the United States, a study was initiated in 1970 to sample 400 sites in 12 States. The sampling areas followed the historical boundaries of the Corn Belt and were selected from sites designated for the National Soils Monitoring Program. At each site a 2-qt soil sample (composite of 50, 2- by 3-inch cores, taken in a grid pattern over each 10-acre site) was collected as well as a composite sample of any available standing crop. In addition, use records were obtained at each site for the kinds and amounts of pesticides used during the 1970 cropping season as well as the names of other pesticides known to have been used in

the previous 5 years. This data indicated that pesticides had been applied to most of the agricultural acreages in the study area (up to 85%). Forty compounds were identified in use records: 20 herbicides, 17 insecticides, and 3 fungicides. Atrazine was most widely used, followed by captan, malathion, 2,4-D, propachlor (Ramrod), amiben, and aldrin. Forty-five percent of the soil samples analyzed contained residues; 11 pesticides or metabolites were detected. Arsenic, which can occur naturally in soil, was detected in nearly all soil samples. The most commonly detected residues were those of aldrin, chlordane, and dieldrin. Seven compounds, including four DDT metabolites, were detected in cornstalks, soybeans, sorghum grain, sorghum fodder, and hay. (Author abstract by permission)

73-2105. Keil, J. E.; Loadholt, C. B.; Sandifer, S. H.; Sitterly, W. R.; Brown, B. L. (Sect. of Preventive Med., Med. Univ. of S. Carolina, Charleston, SC 29401). **Decay of parathion on field-treated tobacco, South Carolina–1972(II).** *Pestic. Monit. J. 6(4): 377-378; 1973.* (2 references)

In an effort to confirm the results of a study in 1971 to determine the length of time required for parathion to degrade to "zero" levels, parathion was applied twice at a rate of .375 lb/acre to field tobacco in South Carolina. After each application, parathion degraded to "zero" levels in five days. These results tended to confirm the findings of the original study in which the maximum time required for parathion to degrade to zero levels was estimated to be seven days and the minimum time two days. Weather was characterized by scanty rainfall and temperatures averaging 76° F. During the original study, rainfall was heavy and daily temperatures averaged 80.9° F. (Author abstract by permission)

73-2106. Baig, M. M. H.; Khan, M. S.; Khan, N.; Ferhat, S.; Hossain, A.; Osmani, M. J. A.; Lord, K. A. (Toxicol. Pesticides Lab., Dept. Plant Protect., Karachi, Pakistan). **Chromatographic and radiometric study of the behaviour of C^{14}-DDT on mustard plants under tropical conditions.** *Pak. J. Sci. Ind. Res. 15(3): 220-226; 1972.* (12 references)

Chemical and radiochemical techniques were used to assess the behavior of a DDT emulsifiable concentrate sprayed onto mustard plants under tropical conditions. DDT, measured as surface radioactivity, was steadily lost from the surface of leaves. This loss rate was similar to that assessed by washing the leaves with hexane and measuring it by gas chromatography and radiometry. About one fourth of the quantity applied remained on leaf surfaces two days after spraying, and less than one tenth remained after ten days. Most of the loss was due to evaporation. After 2-4 days, about one fourth of the DDT had penetrated into leaves, as shown by both chemical and radiochemical measurements of insecticide extractable from leaves after washing the surfaces with hexane. The quantity of DDT in the plant diminished with time, although radiometric assay indicated a faster loss than chemical assay. Most of the radioactivity present in the plant appeared to be in the form of DDT, although gas chromatography gave evidence of slight degradation of DDT.

73-2107. Hill, E. G.; Fishwick, F. B.; Thompson, R. H. (Min. of Agr., Fish., and Food, Pest Infestation Control Lab., Slough, Bucks, England). **Pesticide residues in foodstuffs in Great Britain: organochlorine residues in imported cereals, nuts, pulses, and animal foodstuffs.** *Pestic. Sci. 4(1): 33-39; 1973.* (2 references)

Results are presented for the determination of organochlorine insecticide residues in imported cereals, nuts, pulses, and animal foodstuffs for the 2 year period July 1969 to June 1971. BHC was found in all but four of the 248 samples examined but only in 25 (10%) did the level exceed 0.5 mg/kg. DDT was found in 39% of the samples of which three samples of wheat contained more than 1 mg/kg and two samples of cottonseed cake more than 0.5 mg/kg. The other commodities sampled had less than 0.2 mg/kg. DDE was detected in trace amounts in 26% of the samples examined. (Author abstract by permission)

73-2108. Hill, E. G.; Thompson, R. H. (Min. of Agr., Fish., and Food, Pest Infestation Control Lab., Slough, Bucks, England). **Pesticide residues in foodstuffs in Great Britain: a further report on the bromide contents of imported foods and feedingstuffs.** *Pestic. Sci. 4(1): 41-49; 1973.* (7 references)

Results are presented for the determination of total bromide residues in cereals and cereal products, pulses, oilseed products, dried fruits, nuts, and a variety of other products sampled in Britain during the 3-year period July 1968 to June 1971. Of the 948 samples examined 801 (84%) contained not more than 100 mg/kg bromide while only 20 samples (2%) contained more than 200 mg/kg bromide. The highest residues were found in samples of nuts from India, South Africa, and some East African countries and in some animal feedingstuffs. (Author abstract by permission)

73-2109. Archer, T. E. (Dept. of Environ. Toxicol., Univ. of California, Davis, CA 95616). **Endosulfan residues on alfalfa hay exposed to drying by sunlight, ultraviolet light and air.** *Pestic. Sci. 4(1): 59-68; 1973.* (8 references)

Endosulfan residues on alfalfa hay exposed to drying by sunlight, ultraviolet light, and air under controlled conditions were investigated using GLC and TLC. Maximum loss of total endosulfan and related residues calculated as endosulfan occurred approximately seven days after application and exposure on Lot I (dark) and five days on Lot II (ultraviolet light) and six days on Lot

III (sunlight). Maximum losses of residues on Lot I, Lot II, and Lot III were 48.3, 66.7, and 81.9%, respectively. No endosulfan lactone was detected in any of the samples exposed to the different light treatments. In all lots the endosulfan sulfate percentage of the total residue increased but most dramatically in the dark drying experiment. The endosulfan α-hydroxy ether residues increased in Lot III (sunlight) up to day seven. (Author abstract by permission)

73-2110. Archer, T. E.; Hsieh, D. P. H. (Dept. of Environ. Toxicol., Univ. of California, Davis, CA 95616). **Detoxication of metal drums from emulsifiable concentrate formulations of parathion.** *Pestic. Sci. 4(1): 69-76; 1973.* (5 references)

Metal drums of various sizes which had contained a parathion emulsifiable concentrate were found to retain significant amounts of residues after the containers were completely drained. The residual pesticide formulation was removed from the emptied containers by washing with water and organic solvents. Efficient removal of the parathion by water followed by washing with a water-miscible organic solvent has been demonstrated. It is possible that these washings could be disposed of by combining them into the spray mixture. These procedures suggest a simple and economical way of container decontamination. (Author abstract by permission)

73-2111. Kiigemagi, U.; Wellman, D.; Cooley, E. J.; Terriere, L. C. (Dept. of Agr. Chem., Oregon State Univ., Corvallis, OR). **Residues of the insecticides phorate and methomyl in mint hay and oil.** *Pestic. Sci. 4(1): 89-99; 1973.* (10 references)

Since mint oil is used as a flavoring agent for foods and cosmetics, pesticide residues in the oil are undesirable. The two pesticides, phorate and methomyl, were studied as replacements for chlorinated hydrocarbon pesticides which leave residues in the oil. Phorate applications of 1.1 kg/ha resulted in oil residues of 0.24 mg/kg total phorate in Oregon samples and 1.69 mg/kg in Washington samples. Less than 1% of the residues in the hay distilled with the oil. Mint oil undergoes a 100-to 1000-fold dilution in commercial use with a corresponding reduction of residues in the final product. No residues of methomyl were found in the oil, even with hay residues as high as 5.34 mg/kg. Residues in hay decreased to half of initial levels in 2 to 4 days, independently of use conditions. It was concluded that the proposed use of phorate or methomyl for control of mint pests does not present a residue hazard. Details of the analytical methods used are also presented. (Author abstract by permission)

73-2112. Collins, R. F. (Res. Lab., May and Baker Ltd, Dagenham, Essex, England RM10 7XS). **Perfusion studies with bromoxynil octanoate in soil.** *Pestic. Sci. 4(2): 181-192; 1973.* (7 references)

The breakdown of bromoxynil octanoate in five different soil types has been studied in a soil perfusion apparatus using herbicide labeled with ^{14}C either in the cyano group or in the aromatic ring. Even when applied at rates equivalent to 5 to 25 times those used commercially, the herbicide was fairly rapidly and extensively degraded at 15°. After 12 to 13 weeks up to 80% of the radioactivity in the ^{14}CN group and up to 63% of the ^{14}C in the ring were liberated as carbon dioxide. A small proportion (16 to 19%) of the radioactivity from ring-labeled herbicide remained attached to the soil, probably not as the original herbicide, but in a form not readily leached. Only trace quantities of 2,5-dibromo-4-hydroxy-benzamide (0.5%) and 3,5-dibromo-4-hydroxy-benzoic acid (0.1%) were detectable during these soil perfusions. (Author abstract by permission)

73-2113. Burns, I. G.; Hayes, M. H. B.; Stacey, M. (Chem. Dept., Univ. of Birmingham, Edgbaston, Birmingham, England BI5 2TT). **Spectroscopic studies on the mechanisms of adsorption of paraquat by humic acid and model compounds.** *Pestic. Sci. 4(2): 201-209; 1973.* (23 references)

Changes in the relative sizes of the carboxylic absorption bands in the infrared spectra (near 1730 and 1610 cm^{-1}) of adsorbents before and after treatment with paraquat (1,1′-dimethyl-4,4′-bipyridylium dichloride) showed that ion exchange is the predominant mechanism for adsorption of the bipyridyl by H^1-ion saturated preparations of a humic acid from an organic soil, by two hydroquinone polymers and by two carboxylic ion-exchange resins. Attempts to relate the extents of saturation (with adsorbate) of the exchange sites with the optical densities of the infrared bands were unsuccessful because of deviations from Beer's Law. Ultraviolet spectroscopy failed to provide evidence for charge transfer mechanisms in paraquat–humic acid complexes in aqueous systems. (Author abstract by permission)

73-2114. Smith, D. C.; Leduc, R.; Charbonneau, C. (Health Protect. Branch, Dept. of Nat. Health and Welfare, Tunney's Pasture, Ottawa, Ontario, Canada). **Pesticide residues in the total diet in Canada. III–1971.** *Pestic. Sci. 4(2): 211-214; 1973.* (3 references)

Foods representing an average Canadian diet were cooked, grouped by type of product, and made into composites which were then analyzed for organochlorine and organophosphorus pesticides and arsenic. The amounts found were at a consistently low level and would contribute approximately 22μg total pesticide residues and 25μg arsenic/person/day. (Author abstract by permission)

73-2115. Osgerby, J. M. (Shell Res. Ltd., Woodstock Agr. Res. Cent., Sittingbourne, Kent, England). **Pro-**

cesses affecting herbicide action in soil. *Pestic. Sci. 4(2): 247-258; 1973.* (34 references)

The main processes which affect herbicide action in soil are considered to be the extent to which the herbicide is adsorbed by soil particles, the rate of absorption of the herbicide solution by plant roots, and the rate at which the herbicide is lost from the soil by decomposition, evaporation, or leaching. An attempt is made to show how these factors are interrelated, and the possible use of mathematical models to optimize the performance of herbicides is briefly discussed. (Author abstract by permission)

73-2116. Fadl, F.; Foucault, B.; Rapilly, F.; Leroux, P. (Station Centrale de Pathologie Vegetale, Versailles, France.) **Les traitements fongicides appliques sur le ble:—leur influence sur la composition de la mycoflore du grain—recherche de residus. [Fungicidal treatment of wheat: its influence on the grain mycoflora, residue determinations.]** *Phytiat. Phytopharm. 21(5): 15-23; 1972.* (7 references) (French)

The effect of benomyl on the grain mycoflora of wheat grains, and benomyl residue contents in grains, straw, and husks were studied. Benomyl was applied at a rate of 1.5 kg/ha at the end of anthesis, and the treatment was in some cases repeated 15 and 20 days after the first application. *Alternaria* + *Stemphyllium* counts increased and *Epicoccum* was nearly totally eliminated especially after repeated applications. Quantitative metabolism of benomyl into methyl-2-benzimidazole carbamate in grains, straw, and husks was evidenced. The methyl-2-benzimidazole carbamate residue contents in straw, husks, and grains, determined by thin-layer chromatography on silicagel, were 0.2-16 ppm, 0.1-22.5 ppm, and 0.02-0.50 ppm, respectively. The waiting periods from the date of benomyl application and harvesting ranged from 45 to 65 days. Grains from plants treated three times contained 0.40 ppm of the metabolite.

73-2117. Graham-Bryce, I. J. (Rothamsted Exp. Sta., Harpenden, Herts., England). **Solutions to problems of soil pollution by agricultural chemicals.** *IN: Pollution: Engineering & Scientific Solutions, E. S. Barrekette, ed., Plenum Press, New York-London, 1973, pp.133-147.* (43 references)

The problem of soil pollution by agricultural chemicals primarily involves persistent chemicals like the organochlorines and heavy metals, but some herbicides and fertilizers can also contribute. In general, chemicals are more readily evaporated and photochemically degraded when they are on the soil surface than when incorporated into the soil. Cultivation as a means of decontamination should be limited to soils with incorporated chemical. Addition of adsorbents such as activated carbon may have some occasional usefulness when large quantities of herbicide are required for non-recurrent control of a resistant weed. Increasing general microbial activity by the addition of sugars or various composts or by raising the temperature or maintaining a moist condition is one of the best ways to accelerate biological degradation. Replacement of persistent chemicals by alternative methods of pest control or by less persistent, more specific chemicals is frequently a better approach than employing the above mentioned methods for decontamination of the soil. Care must be exercised, however, in the introduction of biological controls as well. More efficient use of pesticides can be accomplished by application only to the target site, use of granular rather than emulsion formulations, microencapsulation, and exploitation of insect behavior.

73-2118. MacCrum, J. M. (Author address not given). **Operation lake rescue.** *Proc. Inst. Environ. Sci. Annu. Tech. Meet. 18: 238-240; 1972.*

Local, state, and federal agencies worked efficiently together to decontaminate a 5.4-A. lake intentionally poisoned with 1.8 lbs of endrin. The lake, located in Shawnee State Park in Ohio, was contained and the influent was diverted by means of an aluminum pipe. After the poison was identified, activated carbon was distributed on the surface with the hope that adsorption would take place as the material sank to the bottom, but insufficient contact time was achieved. Filling the spillway below the lake with some activated carbon did remove endrin from seepage water, and a successful system of pumping the lake water through a tank containing activated carbon was devised. When the lake was completely drained, plants, which had absorbed endrin, and several inches of bottom sediment were removed.

73-2119. Anonymous. **Chapter 4. Pesticides and toxic substances.** *IN: Report of the Government Chemist 1970. Her Majesty's Stationery Office, London, 1971, pp. 81-103.* (49 references)

The Laboratory of the Government Chemist continues to have an active interest in pesticide residues. Selective studies of organochlorine residues in butter, mutton fat, beef fat, and other items have been made since 1970. Australian and New Zealand butter and mutton fat samples showed a decline in DDT, and the mean dieldrin level of mutton fat was < 0.01 mg/kg, reflecting restrictions on use of these compounds. In short-term residue studies DDT levels found in cod from the Baltic Sea were an order of magnitude greater than those found in cod taken in the North Sea or off the North Cape of Iceland. Mercury levels ranged from 0.02 to 0.07 mg/kg, but did not obviously relate to the place of origin. Organochlorines were not found in citrus fruits from southern Africa, but malathion and parathion were detected, mostly in the outer layer of the peel. Levels of individual organochlorine pesticides in animal feeds seldom exceeded a few hundredths of a mg/kg. Results of a total diet study and a survey of organochlorine

residues in human fat have not yet been compiled. The concentrations of DDT and dieldrin in London rainwater, monitored from 1968 to 1970, have fallen since 1965, and some of the peaks interfering with organochlorine analysis in the rainwater have been tentatively identified as polychlorinated biphenyls. The group developing new methods has concentrated on carbamate insecticides, mercury, the selective flame-photometric detector for GC of organophosphates, and methods for determining residues of veterinary prophylactics. Work has also continued on toxic substances in industrial atmospheres.

73-2120. Mitsuharu, T.; Otsuki, K.; Sekita, H.; Tanabe, H.; Okajima, S.; Sakai, Y. (Nat. Inst. of Hyg. Sci., Tokyo, Japan). **[Studies on analysis of pesticide residues in foods (IX). Effect of cooking on removal of organochlorine pesticide residues from rice, red beans, and soy beans.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 14(2): 142-148; 1973.* (13 references) (Japanese)

Residue standards of 11 pesticides have been officially established on 25 kinds of raw vegetables and fruits in the market. Before served for eating, they will be at least washed, or peeled or cooked. The effect of cooking, especially boiling, on the removal of organochlorine pesticide residues from polished rice, red beans, and soy beans was examined. The specimens are products of 1970, purchased from market. The result shows that a little part of organochlorine pesticide residues including α, β, and γ-BHC, p,p'-DDT and p,p'-DDE was removed from polished rice and red beans, while a considerable part of the pesticide residues was removed from soybeans by boiling. Before washing and cooking, the residual amounts of the pesticide in/on the polished rice were, on the average, 0.003, 0.001, 0.002, 0.010, and 0.005, respectively. After cooking the amounts became, on the average, 0.001, 0.001, 0.002, 0.005, and 0.003 ppm. The residual amounts on/in soy beans before and after cooking and the percentage removal were, on the average, 31, 43, 67, 86, and 20%.

73-2121. Suzuki, M.; Yamato, Y.; Watanabe, T. (Kitakyushu Municipal Inst. of Public Health, Kita-kyushu, Japan). **[On the translocation of soil residual organochlorine pesticides into vegetables.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 14(2): 160-166; 1973.* (5 references) (Japanese)

Several kinds of nearly ripe vegetables including Japanese radish (roots and tops), carrot (roots), turnip (roots and tops), cabbage, spinach, egg plant, cucumber, tomato, and Chinese cabbage, and the soils around the base of the vegetables on which organochlorine pesticides had been applied two to several years ago were collected. Each specimen was analyzed for pesticide residues such as α, β, γ and δ-BHC, aldrin, dieldrin, endrin, and DDT to obtain the information concerning the translocation of pesticide residues in the soils into the vegetables. Each residual BHC-isomer in soil translocated more easily into carrots, turnip tops, Japanese radish tops, and spinach than tomatoes and cabbages. Considering the absolute value of the residue in soil (max. 0.16, 0.8, 0.1, and 0.14 respectively), it may be possible that the residue in carrot and turnip top exceeds the Japanese residue standards. The translocation ratios of aldrin plus dieldrin into cucumbers, carrots, and spinach were 16, 9, and 4, respectively. The translocation ratios of endrin into cucumbers, cabbages, and Japanese radish were 21, 14, and 9, respectively. Considering the residue levels (max. 0.64 ppm) in the soil and the translocation ratios of endrin and aldrin plus dieldrin into the vegetables, there are many possibilities that their residue levels in some vegetables might exceed the Japanese residue standard. Although DDT residue groups in the soils were: p,p'-DDT 9.8-0.01, p,p'-TDE 1.13- 0.01, p,p'-DDE 2.94-0.09, and o,p'-DDT 1.6-0.05 ppm, very little was detected in the vegetables, suggesting little translocation from soil into vegetables.

73-2122. Hiroshima Prefectural Hygiene Research Institute and Hiroshima Prefectural Environmental Protection Research Institute (Hiroshima, Japan). **[Pesticide residue.]** *Showa 46 Nendo Gyomu Nenpo (1971 Annual Report Hiroshima Prefecture) 46: 75-85; 1972.* (Japanese)

From the studies conducted by the Hiroshima Prefectural Environmental Protection Research Institute and the Hiroshima Prefectural Hygiene Research Institute various pesticide residues in soil, fruits and vegetables, milk, processed fish, and human milk are presented in tables. The average residue content was lower in the soil of wet rice fields than in dry vegetable fields. Among the 67 samples of vegetables three samples of potatoes and four samples of cucumbers had dieldrin residues above the level of the maximum allowable residue standard. Twenty-seven samples of cow's milk showed a general decreasing tendency in BHC residues compared to the 1970 measurements. Samples of dried fish had almost no residue of BHC, but small amounts of DDT and DDE. BHC and DDT residues in the farming mothers and non-farming mothers' milk were not significantly different, but the amount of heptachlor epoxide was higher among the farming women.

73-2123. Domanski, J. J.; Laws, J. M.; Haire, P. L.; Sheets, T. J. (Pestic. Residue Res. Lab., N. Carolina State Univ., Raleigh, NC). **Insecticide residues on 1971 U.S. tobacco products.** *Tob. Sci. 17: 80-81; 1973.* (8 references)

Products collected in 1971 containing tobacco produced primarily in 1969 or before contained DDT plus TDE residues similar to those observed during the preceding five years. Residues were about 40 ppm in cigarettes, 22 ppm in pipe tobacco, 18 ppm in little cigars, 16 ppm in snuff, and 13 ppm in chewing tobacco.

Endrin levels dropped in cigarettes and remained the same in snuff. Residues were slightly higher (0.13 ppm) in pipe and chewing tobacco. Toxaphene residues averaged 3.3 ppm, much higher than those observed in the previous year, although its use has been discouraged since 1952. Endosulfan residues averaged 0.25 ppm and probably do not yet reflect the increased use of this insecticide which followed the ban on DDT.

73-2124. Tottori Prefectural Hygiene Research Institute (Tottori, Japan). **[Tests and investigations of pesticide residue in food.]** *Tottori-Ken Eisei Kenkyusho-Ho (Rep. Tottori Pref. Hyg. Res. Inst.) 11: 15-16; 1971.* (Japanese)

A total of 162 samples of cucumbers, tomatoes, grapes, apples, cabbages, Japanese tea, strawberries, Japanese grapefruits, pears, potatoes, spinach, peaches, rice, and eggplants was analyzed for arsenic, lead, gamma-BHC, *p,p'*-DDT, aldrin, dieldrin, endrin, parathion, and EPN residues (also cadmium and mercury in rice). Three pear samples contained more than the maximum allowable amount of DDT (1.191, 1.683, and 1.733 ppm), and one sample each of potato and spinach had dieldrin residues (0.0075 and 0.0083 ppm respectively). The rest had no significant trace of residues.

73-2125. Tottori Prefectural Hygiene Research Institute (Tottori, Japan). **[Tests and investigations on pesticide residues in milk and dairy products.]** *Tottori-Ken Eisei Kenkyusho-Ho (Rep. Tottori Pref. Hyg. Res. Inst.) 11: 17-18; 1971.* (Japanese)

Fifty-nine samples of milk and 16 samples of dairy products in Tottori Prefecture are regularly analyzed for organochlorine pesticide residues (alpha, beta, gamma, delta-BHC, *p'*-DDT *p,p'*-DDT, *p,p'*-DDE, *p,p'*-DDD, aldrin, dieldrin, and endrin) every other month. In 1971 inspections all samples except for one case each of whole milk and condensed milk had less than the maximum allowable residue standard. Dieldrin above the WHO levels was detected in one sample each of milk and condensed milk, and there was no trace of aldrin, endrin, and *o,p'*-DDT. However, all the processed dairy products had some traceable amount of dieldrin, which could cause a chronic effect. The amount ranged from 0.0133 to 0.1254 ppm (average 0.0720) in butter and 0.0035 to 0.3115 ppm (average 0.0881) in condensed milk.

73-2126. Tottori Prefectural Hygiene Research Institute (Tottori, Japan). **[Tests and investigations on pesticide residues in mother's milk and blood.]** *Tottori-Ken Eisei Kenkyusho-Ho (Rep. Tottori Pref. Hyg. Res. Inst.) 11: 19; 1971.* (Japanese)

Twenty samples each of mother's milk and blood were analyzed for pesticide residues in Tottori Prefecture. Samples were taken from 10 women from farming families (group I) and 10 from non-farming families (group II). There was a clear and notable amount of residue in mother's milk. Beta BHC ranged from 0.037 to 0.296 ppm for I, and 0.028 to 0.174 for II; gamma BHC ranged from 0.005 to 0.0084 for I and 0.0006 to 0.0057 for II; total DDT ranged from 0.0180 to 0.3047 for I and 0.0390 to 0.2139 for II; dieldrin ranged from zero to 0.0234 for I and zero to 0.0048 for II. The number of samples exceeding the maximum allowable standards were 2, 1, 8, and 8, in group I, and 8 for total DDT in group II. No significant residue was found in blood.

73-2127. Tottori Prefectural Hygiene Research Institute (Tottori, Japan). **[Tests and investigations on pesticide residues in food.]** *Tottori-Ken Eisei Kenkyusho-Ho (Rep. Tottori Pref. Hyg. Res. Inst.) 12: 16-18; 1972.* (Japanese)

A total of 158 samples of strawberries, cabbages, potatoes, tomatoes, apples, cucumbers, grapes, peaches, pears, Japanese tea, spinach, turnips, and rice was analyzed this year for residues of arsenic, lead, gamma-BHC, *p,p'*-DDT, aldrin, dieldrin, endrin, parathion, and EPN (also cadmium in rice). Compared to last year's record, almost no trace of DDT was detected in pears, and generally BHC residue levels were lower, showing the effect of stringent enforcement of agricultural chemical regulations in the prefecture. However, some dieldrin and endrin residue was found in vegetables. Those that failed to pass the standard were 0.014 ppm and 0.006 of endrin in turnip root and leaves, 0.018 ppm and 0.035 ppm of dieldrin in potatoes and cucumbers, respectively.

73-2128. Tottori Prefectural Hygiene Research Institute (Tottori, Japan). **[Tests and investigations on pesticide residue in cow's milk.]** *Tottori-Ken Eisei Kenkyusho-Ho (Rep. Tottori Pref. Hyg. Res. Inst.) 12: 19-21; 1972.* (Japanese)

Forty samples of milk and 35 samples of dairy products (butter, condensed milk, cheese, powdered milk) were analyzed in 1972 in Tottori Prefecture for residues of organochlorine pesticides. All the samples had less-than-standard residues, but there was no marked over-all improvement in traceable residue levels. The pesticide regulations of the prefecture have not shown much effect in milk and dairy products. However, powdered milk for babies (9 samples) had a commendably small amount of residue. Average residues in powdered milk were 0.071 ppm of α-BHC, 0.113 ppm of β-BHC, 0.012 ppm of γ-BHC, 0.004 ppm of δ-BHC, 0.062 ppm of *p,p'*-DDE, 0.026 ppm of *p,p'*-DDT, 0.025 ppm of *p,p'*-DDD (TDE), and 0.013 ppm of dieldrin. Aldrin, endrin, and *o,p'*-DDT were not detected in any sample.

73-2129. Tottori Prefectural Hygiene Research Institute (Tottori, Japan). **[Tests and investigations of pesticide**

residue in mother's milk. | *Tottori-Ken Eisei Kenkyusho-Ho (Rep. Tottori Pref. Hyg. Res. Inst.) 12: 22; 1972.* (Japanese)

In an inspection program continued since last year 10 samples of human milk (five farming women, five non-farming women) were analyzed in Tottori Prefecture for organochlorine pesticide residue. Three samples of non-farming women and four samples of farming women exceeded the maximum allowable residue standard of dieldrin. The highest count in each category came from samples of farming women in all cases. Counts were β-BHC 0.108 ppm, γ-BHC 0.0013 ppm, total DDT 0.124 ppm, dieldrin 0.0047 ppm, and heptachlor 0.0010 ppm.

See also

73-2034	73-2133	73-2147	73-2176	73-2225	73-2254	73-2265
73-2049	73-2135	73-2175	73-2224	73-2232	73-2261	73-2276
73-2050	73-2143					

73-2130. Davies, J. E. (Dept. Med., Univ. Miami School Med., Miami, FL). **Recognition and management of pesticide toxicity.** *Advan. Intern. Med. 18: 23-38; 1973.* (65 references)

Classes of pesticides with known diagnostic and therapeutic difficulties are reviewed in relation to information which should be a part of the physician's background. Organophosphates, especially parathion, are involved in human poisonings more frequently than any other pesticide class. Treatment must be initiated on the basis of clinical diagnosis alone since laboratory tests used for confirmation require more time than is available. Muscarinic and nicotinic effects are signs of systemic poisoning, and death is generally due to respiratory failure. Restoration of proper breathing and correction of hypoxia, atropine and oxime therapy, decontamination, and identification of the source of the poison to prevent future accidents are measures which generally should be taken. Paraquat, a bipyridyl causing proliferative changes in the lung, cornea, lens, nasal mucosa, skin, and fingernails, is generally fatal if ingested. Treatment includes attempts to limit the amount of material absorbed through gastric lavage and administration of Fuller's Earth and forced diuresis. Poisoning from organochlorines is generally the result of ingestion of a concentrate and is characterized by central nervous system stimulation. Drugs such as barbiturates should be administered as quickly as possible to initiate CNS sedation. Residues of organochlorines in blood and fat have been used to estimate length and severity of exposure.

73-2131. Anonymous. **Safe disposal for pesticide containers.** *Agr. Res. 21(8): 8-9; 1973.*

Thermal disposal (incineration) seems to be the best method immediately available for disposing of pesticide containers. Not only the containers, but also the pesticide remaining in them (about 2.5% of the original contents) must be eliminated. In the Mississippi study researchers determined that temperatures around 1,000°C would degrade 99% of most current pesticidal formulations. All but metal containers are reduced to ash at this temperature. A model incinerator, which has multiple combustion chambers and can handle liquids and solids, is presently being studied. Ultimately, this technique will probably be combined with chemical treatment and microbial degradation.

73-2132. Snyder, N. F. R.; Snyder, H. A.; Lincer, J. L.; Reynolds, R. T. (Box 21, Palmer, Puerto Rico 00721). **Organochlorines, heavy metals, and the biology of North American accipiters.** *BioScience 23(5): 300-305; 1973.* (14 references)

Population declines have been observed since 1947 in Cooper's and sharp-shinned hawks in Pennsylvania-New York; in a 3-yr study Oregon Cooper's hawk and goshawk populations remained stable, while numbers of sharp-shinned hawks fluctuated. In Arizona-New Mexico the population of Cooper's hawk has been stable from 1969 to 1971 and numbers of goshawks and sharp-shinned hawks are too small to indicate a trend. A significant correlation was observed with birds from all regions between eggshell thinning and DDE content, and DDE seemed to be associated with egg breakage rather than infertility or embryonic death. A sufficient number of less contaminated eggs have been produced in the West to maintain the population. The Cooper's hawk and the sharp-shinned hawk, higher in the food chain than the goshawk, were most noticeably contaminated with DDE. In Arizona-New Mexico abnormal behavior was observed in 3 of 11 pairs of adult Cooper's hawks studied, and high levels of DDE were found in the chicks and eggs of these birds. Levels of mercury, lead, cadmium, copper, dieldrin and polychlorinated biphenyls in eggs taken in Arizona-New Mexico were low and generally did not correlate with eggshell thinning and reproductive success.

73-2133. Newton, I. (Nature Conservancy, 12 Hope Terrace, Edinburgh 10, Scotland). **Success of sparrowhawks in an area of pesticide usage.** *Bird Study 29(2): 1-8; 1972.* (13 references)

Sparrowhawk nesting sites checked over the period from 1967 to 1971 indicated an approximate doubling in numbers of these birds in the study area of southern Scotland. However, extensive observations made in 1971 showed no improvement in breeding success. Of the 113 occupied sites, 93 had nests lined and laid in. In 69 of these at least one young hatched and in 66 at least one young fledged. Most of the losses resulted from failure to lay eggs or failure to hatch them. Circumstantial evidence showed that the hen herself was responsible for some egg breakage. Egg-shell thinness, determined by the weight/size ratio, probably caused additional breakage. Between 96 and 711 ppm organochlorine residues were found in the lipid of the ten eggs tested. These residues could be responsible for the 56% reduction in the potential output of young in the Sparrowhawk population.

73-2134. Bradfield, P. L. (Michigan State Univ., East Lansing, MI). **Robin population changes and pesticide transformations after methoxychlor replaces DDT for control of Dutch elm disease.** *Diss. Abstr. Int. 33(9): 4238B; 1973.*

After Michigan State University changed from DDT to methoxychlor in 1964 to protect campus elms for Dutch elm disease, robin (*Turdus migratorius*) mortality decreased but continued for six years due to the persistence of DDT in the earthworms. Studies were made of the residues of DDT, DDD, DDE, and methoxychlor in the soil, earthworms, and robins. Techniques for contamination of the nightcrawler (*Lumbricus terrestris*) with known amounts of DDT were perfected. Night-

crawlers with known amounts of DDT and methoxychlor were fed to robins and red-winged blackbirds (*Agelaius phoeniceus*) to determine the lethal threshold of DDT and methoxychlor. Brain and body tissues of experimental birds were analyzed so that pesticide levels in birds dying in the field would be helpful in diagnosing the cause of death. Field studies and laboratory results both indicate that some robins will die when eating earthworms containing 20 to 30 ppm DDT plus DDD. Worms from soils containing four or more ppm DDT plus DDD can obtain the necessary 20 plus ppm to kill robins. All laboratory work indicates that methoxychlor in the amounts found in the field, and in even greater amounts, had no observed effect on robins and is accumulated at less than five percent of the rate of DDT, by birds. Robins and red-winged blackbirds are similar in their response to DDT and methoxychlor but vary widely in their food preferences. (Author abstract by permission. Copies of the thesis are available from University Microfilms, Order No. 73-5333.)

73-2135. Lincer, J. L. (Cornell Univ., Ithaca, NY). **The effects of organochlorines on the American kestrel (*Falco sparverius* Linn.)** *Diss. Abstr. Int. 33(9): 4240B-4241B; 1973.*

DDE residues and eggshell thickness were examined in eggs from a captive kestrel colony fed diets containing DDE and/or Aroclor 1254 and in wild kestrel eggs collected around Ithaca, New York. DDE residues in wild kestrel eggs averaged 35, 42, and 33 ppm, oven-dry basis, for 1969, 1970, and 1971, respectively. Eggs collected during 1970 contained 37 ppm PCB, almost 3 ppm dieldrin, less than 1 ppm TDE and DDT, and less than 0.5 ppm mercury, copper, cadmium, or lead. Eggshells of the local kestrel population averaged 9% thinner than eggshells collected before 1947. An inverse statistical correlation was found between DDE residues in wild kestrel eggs and the eggshell thickness. Dietary DDE levels of 0.3 to 10 ppm result in egg DDE levels of about 2.1-70 ppm. The relationship between egg DDE residues and eggshell thinning in the experimental birds is biphasic, showing an initial sharp decrease in shell thickness correlated with low egg residues. The statistical relationship between eggshell thickness and egg DDE level was similar to that in the wild kestrels. PCBs apparently synergize the eggshell thinning effect of DDE. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-5857.)

73-2136. Drayer, D. E.; Reidenberg, M. M. (Dept. Pharmacol., Temple Univ. School Med., Philadelphia, PA 19140). **Metabolism of tetralin and toxicity of Cuprex in man.** *Drug Metab. Disposition 1(3): 577-579; 1973.* (8 references)

Two nonconjugated metabolites and unchanged tetralin (1,2,3,4-tetrahydronaphthalene) were found in the urine of a woman who intentionally ingested one-half to three-fourths of a pint of Cuprex. One metabolite was identified as 1,2,3,4-tetrahydro-1-naphthol (I), and mass spectra of the second metabolite indicate that it was quite similar, but identification was not made. The glucuronides of the tetralin metabolites were also found in the urine. Liver disease in the patient could have been responsible for the excretion of some nonconjugated metabolites since healthy rabbits excreted only the glucuronides. The patient experienced a mild degree of renal injury and transient liver damage, both of which subsided within 14 days.

73-2137. Cardozo, L. J.; Mugerwa, R. D. (Dept. Med., Makerere Univ. Med. School, Kampala, Uganda). **The pattern of acute poisoning in Uganda.** *East Afr. Med. J. 49(12): 983-988; 1972.* (8 references)

A rising incidence of poisoning with non-drug poisons, especially pesticides, was noted in a survey of the cases admitted during 1970 to Mulago Hospital in Uganda. Pesticides were responsible for the admission of 9 of 48 adults and 10 of 22 children. Self-poisoning was more prevalent among adults. Pediatric admissions were all due to accidental exposure. An equal number of male and female adults were admitted, and the ratio of male : female in children was 1:0.8. One death occurred as a result of pesticide poisoning.

73-2138. Govaerts, M.; Vincent, V.; Jouglard, J.; Efthymiou, M. L. (Author address not given). **Bibliographies des livres de toxicologie utiles dans un centre antipoisons d'information. [Bibliographies of useful books for poison information centers.]** *Eur. J. Toxicol. 5(5): 322-332; 1972.* (French)

A bibliography of books on toxicology containing several hundred entries useful for poison information centers is presented on the basis of lists sent by poison information centers in different European countries. Entries are rated as essential, very useful, recommended for purchase, or not worthwhile. In addition, availability of the books in different poison information centers in Europe is indicated.

73-2139. Kessler, H.; Mracek, J. F. (Pestic. Accident Investigator, Pestic. Branch., U.S. Environ. Protect. Agency, Atlanta, GA 30309). **Nonfatal accidental organophosphate pesticide intoxication in seven inmates of a correctional institution.** *J. Med. Ass. State Ala. 42(11): 775-781; 1973.* (6 references)

Nine prisoners showed symptoms of organophosphate poisoning almost immediately after drinking a homemade beverage which they had unknowingly contaminated with diazinon concentrate. The source of the poison was identified, and the men were treated in the hospital with atropine and 2-PAM. Cholinesterase levels were low in all of the patients, but below normal in only

one, and glucose levels were above normal in all nine of the men. All of the men recovered. Each said he would have consumed the beverage realizing that it was contaminated since the toxicity factor was unknown.

73-2140. Herman, P. S. (Cent. Hospitalier Universitaire, Sherbrooke, P.Q., Canada). **Hexachlorobenzene pesticides and porphyria.** *Med. J. Aust. 1(6): 315; 1973.* (3 references)

In a Letter-to-the-Editor it was suggested that people in the pesticide industry and porphyrin researchers work together to assess the actual toxic potential of hexachlorobenzene (HCB). One author has reported high blood levels in subjects occupationally exposed to HCB and who exhibited no symptoms of toxicity. No information was given concerning the parameters observed. In a report of acquired toxic porphyria cutanea tarda due to hexachlorobenzene no reference was made to blood or fat levels of HCB.

73-2141. Morley, A.; Geary, D.; Harben, F. (Dept. Med., Univ. Adelaide, Adelaide, Australia). **Hexachlorobenzene pesticides and porphyria.** *Med. J. Aust. 1(11): 565; 1973.* (2 references)

A Letter-to-the-Editor reports that one out of 54 workers in a factory which prepared and distributed grain treated with hexachlorobenzene (HCB) had an elevated porphyrin excretion. The man who was 64 years old and had been exposed to HCB for 25 years, excreted 170 μg of uroporphyrin and 255 μg of coproporphyrin in 24 hours in the urine. A qualitative test indicated elevated fecal porphyrin excretion. Since none of the other subjects exhibited the same porphyrin excretion, the one case could have been coincidental. However, other authors have reported a case of porphyria in a worker occupationally exposed to HCB.

73-2142. Simpson, G. R. (Div. Occup. Health Pollut. Contr., Dept. Health, Lidcombe, New South Wales, 2141 Australia). **Aerial spraying of organic phosphate pesticides lowered blood cholinesterase levels of aerial spray operators at Wee Waa.** *Med. J. Aust. 1(15): 735-736; 1973.* (1 reference)

Surveys of blood cholinesterase conducted on a voluntary basis on aerial spray crews in the cotton-growing area at Wee Waa showed that at least 50% of the subjects had ChE values below normal. A number were at a level indicating that further exposure should be avoided and two had already produced symptoms. Parathion-contaminated overalls, spillage of monocrotophos during loading, and accidental contamination of bare hands with mevinphos were responsible for some of the lower levels. Protective clothing was not generally provided by the contractors, and laundering of the clothing was done by the employees. No spare overalls were available in the field in case of contamination, and generally, running water was not provided for hand washing. Human markers were used against the recommendation of the Pilot Manual Instruction for spraying organophosphates. Leaky inboard pesticide pumps and hoppers as well as barehanded handling of the jet plates of spray boom were probably responsible for contamination of pilots.

73-2143. Cress, D. (Author address unknown). **Pesticides Manual: Classification, Toxicities, Formulation, Handling, Application.** *Mich. Coop. Ext. Serv. Ext. Bull. (E-751), Michigan State University, East Lansing, 1972, 23 p.*

In a bulletin describing the classification, characteristics, formulations, compatibilities, and modes of application of pesticides, the residual life of some herbicides, the toxicity ratings of chemicals representing various classes of toxicity, and recommendations for the safe use of pesticides are listed. Residual life of herbicides and other pesticides in soils affects duration of activity of the compounds, unwanted effects on plants and animals, and environmental pollution. The range for herbicides is from 0 to 10 days with paraquat to over 120 days with diuron. All directions must be followed explicity for the application and handling of pesticides. Proper clothing and protection equipment must be worn, and containers should be rinsed three times and drained into the spray tank and then destroyed by burning (except in the case of 2,4-D-type chemicals) or buried deeply where the water supply will not be contaminated. Storage of pesticides should be in a locked area not accessible to children or livestock. The hazards of pesticides to humans vary depending on their use. Four classes of toxicity have been established on the basis of LD50s. Class 1, highly toxic, includes those chemicals with oral LD50s of 1 to 50 mg/kg and dermal LD50s of 1 to 200 mg/kg in rats.

73-2144. Anonymous. **Tabulations of 1971 reports.** *Nat. Clearinghouse Poison Contr. Cent. Bull. 1-9; 1972.*

Data reported for 1971 from 505 poison control centers shows that pesticides were responsible for 6,446 out of 136,051 cases of poisoning. The largest percentage of these cases was 1- or 2-yr-old children. More males than females were reported to have been poisoned by pesticides, and only 49 out of the 6,446 cases were suicide attempts. Some increase has been observed since 1968 in the incidence of poisoning by insecticides and rodenticides.

73-2145. Straughan, R. P. L. (Author address not given). **Pollution, dredging decimating Florida's reefs.** *Nat. Fisherman 52(13): 46-48; 1972.*

Mosquito spray operations, while considered quite detrimental to the smaller reefs, were probably not the major culprit in the death of the famous Hen and

Chickens Reef in Upper Florida Keys. Shrimp and other aquatic invertebrates are extremely susceptible to insecticides in the ppb range, and spray operations have been carried out extensively in the Keys for up to 10 years. However, miles of the Hen and Chickens Reef are covered with a layer of silt from the dredging and filling along the shore. Scientists have shown that a thin layer covering coral for 48 hours is sufficient to kill the organism. The effect of the dredging has gone unnoticed, and plans to fill a reef for an oceanographic laboratory were being carried out until the scientists at a nearby laboratory were notified. Sewage pollution may have also contributed to the death of the reef. Massive efforts to stop the tremendous growth in the area will be necessary to prevent the death of other reefs, including Pennekamp Park.

73-2146. Gilbert, D. (Author address not given). **Did all-ocean alarm make the right waves?** *Nat. Fisherman 52(13): 62-65; 1972.*

In 1971-72 concern over the pollution of the ocean by petroleum was aroused by Thor Heyerdahl, an explorer, and Jacques Cousteau, an oceanographer. Although their interest has alerted many to the dangers involved in misuse of the ocean, a more scientific view of the problem shows that petroleum is biodegradable and that organochlorine pesticides, as well as over-fishing, are more important factors reducing the oceans' productivity. A panel assigned by the National Academy of Sciences reported that organochlorines have been found in plankton, the base of the food chain, and in many aquatic organisms. DDT has been suspected of causing many unexplained fish kills. International agreement on fishing practices, contamination of the water, etc. must be reached in order to preserve the presently increasing annual catches.

73-2147. Herngreen, J. J.; Zielhuis, R. L. (Coronel Lab., Fac. of Med., Univ. of Amsterdam, Holland). **Borstvoeding en bestrijdingsmiddelen. [Breast feeding and pesticides.]** *Ned. Tijdschr. Geneesk. 116(13): 511-518; 1972.* (32 references) (Dutch)

Reports of pesticide contamination in human milk raised the question of whether breast-feeding of infants is advisable from this viewpoint. According to surveys of lactating mothers in the Netherlands and other western countries, a ten-day-old infant may ingest 4 μg/kg body weight/day of DDT, 0.2 μ/kg dieldrin, 3 μg/kg BHC, and 1.5 μg/kg heptachlor epoxide via the mother's milk. These values are respectively 4, 0.8, 0.1, and 0.25 times the acceptable daily intake levels established for adults by the World Health Organization. Levels of these insecticides in cow's milk are appreciably lower except for BHC. Specific effects of organochlorine ingestion which may be significant to the infant include enzyme induction, alteration of growth and development, interference with metabolic processes in the cell membrane, and changes in central nervous system function. The no-effect level of DDT for enzyme induction in the rat was established as 10 μg/kg/day, a value which may be exceeded by the breast-fed infant. Interpretation of these findings is obscured by the paucity of pesticide toxicity studies performed on young animals. However, it does not appear necessary to discourage breast feeding because of pesticide contamination at present.

73-2148. Matsushima, S. (Saku General Hospital, Saku, Nagano Prefect., Japan). **[Problems of agricultural pollution.]** *Nogyo Oyobi Engei (Agr. Hort.) 48(1): 233-238; 1973.* (Japanese)

The number of accidents involving pesticides has remained between 1035 and 1737 per year between 1958 and 1969. Subsequent mortality is greater than 800 every year, most of which (700-800) is from suicide with pesticides. This demonstrates the loose and uncontrolled use of pesticides. Records only show reported and investigated cases. In the Saku area the records of eight communities using chemicals for rice fields, apples, chrysanthemums, and vegetables showed that approximately one-fourth of the total population using pesticides experienced some degree of poisoning during the spray period of June to September. Of the total 649 patients hospitalized with pesticide poisoning from 1967-1968, 38.4% had widespread acute symptoms, 38.4% had skin damages, 17% had eye damages, 2.9% had pneumonia, bronchitis, and bronchial asthma, 1.7% had pharyngitis, laryngitis, and rhinitis, 3.1% had liver trouble, 0.8% had intestinal troubles, and 0.3% had cystitis. Some indiscriminate aerial spray has caused accidents involving hundreds of school children. Residues in soil, river, air, produce, processed food, and the human body are also discussed.

73-2149. Slocombe, G.; Thorn, P. E.; Toohill, J.; Wood, J. (Intensive Care Unit, Princess Margaret Hosp., Swindon, Wilts, England). **A case of paraquat poisoning.** *Nurs. Times 69: 111-112; 1973.* (1 reference)

Following suspected ingestion of a maximum of 2.2 g of paraquat, a 45-yr-old woman was brought to the Intensive Care Unit. No treatment had been administered while confirmation by a rapid screening test was performed, and it was considered too late to perform gastric lavage on admission. Hemodialysis was used for nine days along with intravenous administration of fluids to increase urinary output. Panadol and Valium were administered after complaints of abdominal pain. Piriton was given intramuscularly for a rash that developed on the abdomen and thighs. Upon recommendation of a dermatologist, Valium and Panadol were discontinued. Recovery was probably a result of early treatment since no symptoms of poisoning had been evident when the patient was admitted.

73-2150. Schroeder, M. H. (U.S. Bur. Sport Fish. Wildlife, Denver, CO 80225). **Relationships of range and**

forest management with herbicides on wildlife. *Proc. West. Soc. Weed Sci. 25: 11-14; 1972.* (18 references)

Herbicides can be used beneficially by range and forest managers. Studies have indicated that most of these chemicals present no danger of direct toxicity to man or animals since they degrade within a few weeks. Teratogenic effects were observed with one compound, 2,4,5-T, due to a toxic contaminant, and use of this compound has been restricted. Toxicity to fish has only been observed at rather high concentrations of herbicides in water. Fish have shown an ability to leave water containing high concentrations until dilution takes place. Indirect effects on wildlife have occurred as a result of eutrophication of aquatic environments treated with herbicides and deprivation of food and cover. Preparation of food patches, creation of deer browse, and brush clearing through selective use of herbicides have resulted in increases in desired species. Recreational areas have also been cleared with these pesticides. Both game and nongame wildlife should be considered when control programs are planned.

73-2151. Anonymous. **Oprettelse af en giftinformationscentral. [Establishing a poison information center.]** *Sundhedsstyrelsen 6(6): 65-68; 1972.* (Danish)

Activity of the newly established Poison Information Center located in the Rigshospitalet in Copenhagen and plans for future extension are described. The Poison Information Center presently is manned by a physician and two pharmacists. The number of pharmacists will be increased to four in the near future. Some 60% of all phone calls concerning household chemicals and pesticides were concerned with poisonings of children. The activity of the Poison Information Center will be aided by electronic data processing.

See also

73-2072 **73-2084** **73-2094** **73-2099** **73-2100** **73-2110** **73-2181**

73-2152. Varshney, T. N.; Gaur, A. C. (Div. Microbiol., Indian Agr. Res. Inst., New Delhi, India). **Effect of DDT and Sevin on soil fungi.** *Acta Microbiol. Acad. Sci. Hung. 19: 97-102; 1972.* (27 references)

DDT was more detrimental to soil fungal populations in the laboratory than Sevin (carbaryl), but both insecticides inhibited *Alternaria humicola, Trichoderma viride, Botryotrichum* sp., *Helminthosporium sativum, Sepedonium* sp., and *Papularia* sp. DDT inhibited *Mucor* sp., *Hormiscium* sp., *Stachybotrys atra, Rhizoctonia solani, Synsporium* sp., *Gliocladium sagariensis, Botrytis* sp., *Myrothecium straitisporum, Phoma humicola* and *Macrophoma* sp., species which were stimulated by carbaryl. Farmyard manure seemed to counteract the depressing effect of DDT.

73-2153. Gajduskova, V.; Lat, J. (Vet. Res. Inst., Brno, Czechoslovakia). **Effect of malathion and *N*-methyl-carbamates on growth and morphology of yoghourt culture organisms.** *Acta Vet. Brno 41: 447-452; 1972.* (9 references)

Malathion did not inhibit the growth of yogurt culture organisms when added to milk at concentrations of 20 or 100 ppm. A reduced acid production, a slowed rate of coagulation, and the formation of gross tufts and whey resulted when the level was increased to 20 ppm. Repeated transfers in milk containing 200 ppm malathion caused inhibition of the growth of streptococci and elongation and chaining of lactobacilli. The ratio of streptococcus/lactobacillus stabilized after five transfers. Aprocarb (propoxur), carbaryl, and 1-naphthol inhibited acid production at levels as low as 20 ppm, but coagulation did not appear to be affected. Repeated transfers in milk containing *N*-methyl carbamates resulted in elongation of lactobacilli but did not inhibit the growth of streptococci. At 200 ppm *N*-methyl carbamates led to permanent degeneration of the cells. The results suggest that carbamates excreted in the milk may affect the quality of dairy products.

73-2154. Nunogawa, J. N.; Burbank, N. C.; Young, R. H. F. (Water Resources Res. Cent., Univ. Hawaii, Honolulu, Hawaii). **The relative toxicities of selected chemicals to several species of tropical fish.** *Adv. Water Pollut. Res., Proc. Int. Conf. 5th, 1971, pp. HA-14/1–HA-14/14.* (10 references)

Baseline data on the toxicity of three organochlorines to common tropical fish was obtained. Easily duplicated methods using available equipment indicated that over 96 hr lindane was the least toxic, followed by dieldrin and DDT. Phenol was several orders of magnitude less toxic than the organochlorines. *Gambusia affinis* was the least sensitive of the five fish tested, and *Kuhlia sandvicensis* and *Stolephorus purpureus* were the most sensitive. *Lebistes reticulatus* and *Tilapia mossambica* had a higher sensitivity to phenol. The 24-hr TLm values for DDT ranged from 0.12 to 0.56 mg/l. for DDT, 0.044 to 0.56 mg/l. for lindane, and 0.0059 to 0.085 mg/l. for dieldrin. The results generally agreed with those of other studies.

73-2155. Zabik, M. E.; Schemmel, R. (Dept. of Food Sci. and Human Nutr., Michigan State Univ., East Lansing, MI 48823). **Dieldrin storage of obese, normal, and semistarved rats.** *Arch. Environ. Health 27(1): 25-30; 1973.* (23 references)

Dieldrin tissue levels in obese and normal weight adult male Osborne Mendel rats that had been on weight maintenance or weight reduction diets were compared with levels immediately after dieldrin feeding. Normal weight animals exhibited higher tissue pesticide reductions while very little of the body burden of dieldrin was lost in obese rats. Neither group exhibited any elevation of dieldrin levels in the brain because of tissue mobilization. (Author abstract by permission)

73-2156. Brody, I. A. (Div. Neurol., Duke Univ. Med. Cent., Durham, NC). **Myotonia induced by monocarboxylic aromatic acids. A possible mechanism.** *Arch. Neurol. 28: 243-246; 1973.* (31 references)

The K-independent *p*-nitrophenylphosphatase (basic *p*-NPPase) activity of microsomes isolated from normal rat skeletal muscle was increased by 2,4-D and 3-chloro-2,5,6-trimethylbenzoic acid. The activities of K^+-stimulated *p*-NPPase and Na^+,K^+-stimulated adenosine triphophatase were unchanged. Similar findings were observed in muscle microsomes from rats made myotonic with 2,4-D (200 mg/kg, i.p. injection). Increased basic *p*-NPPase activity seems to be related to increased passive flux of K^+ which might lead to myotonia through a compensatory decrease in Cl-conductance.

73-2157. Medline, A.; Bain, E.; Menon, A. I.; Haberman, H. F. (Dept. Pathol., Toronto Western Hosp., Toronto M5T 2S8, Canada). **Hexachlorobenzene and rat liver.** *Arch. Pathol. 96(1): 61-65; 1973.* (22 references)

Livers of rats fed a diet containing 0.2% hexachlorobenzene showed marked enlargement of the hepatocytes, many of which contained discrete eosinophilic laminated cytoplasmic bodies, by the end of the third week. The laminated intracellular structures, observed under the electron microscope, were fingerprint configurations and myelin-like figures. Other changes resulting from administration of hexachlorobenzene included protrusion of cytoplasm through the space of Disse into the sinusoid, the presence of membrane-bound vacuoles containing organelles and glycogen in the sinusoids, an apparent increase in the smooth endoplasmic reticulum, and formation of lipid droplets. No significant change was observed in the mitochondria.

73-2158. Rosenkranz, H. S. (Dept. Microbiol., College Physicians Surgeons, Columbia Univ., New York, NY 10032). **Preferential effect of dichlorvos (Vapona) on bacteria deficient in DNA polymerase.** *Cancer Res. 33(3): 458-459; 1973.* (22 references)

Escherichia coli deficient in DNA polymerase were more sensitive to dichlorvos than the parents from which they were derived, a characteristic of other DNA-alkylating agents known to be carcinogenic. Substances which do not affect cellular DNA did not inhibit the DNA-deficient strain preferentially. The data suggests that the DNA of living cells is altered by dichlorvos. This has already been demonstrated in vitro. Since this chemical is used widely where human exposure is extensive, a reexamination of the potential hazard to human health is imperative.

73-2159. Nakajima, S.; Naito, N.; Tani, T. (Hoshi College Pharm., 2-Chome, Ebara, Shinagawa, Tokyo, Japan). **Microbial transformation of 2,4-D and its analogues.** *Chem. Pharm. Bull. 21(3): 671-673; 1973.* (6 references)

Three molds, *Glocosporium olivarum, Glocosporium kaki* and *Schizophyllum commune* metabolized 2,4-D, 2,4,5-T and 2-methyl-4-chlorophenoxyacetic acid (MCP) to products having antifungal activity. The products were isolated and identified as the phenoxyethanols of the respective herbicides. Growth indices indicated that their antifungal activity was much greater than that of the parent compound. The fact that the herbicides were reduced in the presence of the molds contradicts the opinion that the herbicidic activity of 2-(2,4-dichlorophenoxy)ethanol on higher plants is operative after oxidation of 2,4-D.

73-2160. Tachibana, H.; Hashimoto, K.; Ishibashi, T.; Ando, T.; Kitagawa, M. (Osaka Prefect., Inst. of Pub. Health, Osaka, Japan). **[Studies of the learned behavior of rats as an index for toxicity.]** *Dai 46-tai Nippon Sangyo Eisei Gakkai Koenshu (Proc. Jap. Ind. Hyg. Soc. Annu. Meeting) 46: 408-409; 1973.* (Japanese)

Carbaryl was given to three groups of rats and its effects on spontaneous actions and avoidance learning examined. Body weight, liver weight, and AChE levels in the brain were also studied. Controls were given the standard feed, and the experimental animals were given carbaryl mixed with feed at doses of 100, 500, and 2500 ppm. Avoidance learning training used a shuttle box with an electric shock, and frequency of failure to avoid the shock was counted every ten minutes. In the beginning the group given 100 ppm carbaryl had less shock frequency than any other group. The frequency of the control group was greatest. As training continued, the record of the control group improved, and the averages for all groups were 1.34, 2.90, 4.97, and 8.24. Both the 100 and 2500 ppm groups experienced slight weight loss after 35 days of the drug. Liver weight seemed to increase in proportion to the amount of carbaryl although the differences were not statistically significant. AChE levels were highest in the control group.

73-2161. Bates, J. J. (Univ. of Missouri, Columbia, MO). **Oxycarboxin: site of action, mode of action, and degradation.** *Diss. Abstr. Int. 33(9): 4067; 1973.*

Studies were performed to determine the site of oxycarboxin action on rust disease of pinto beans, and oxycarboxin degradation in soil, nutrient solution, and plant was examined. Results suggested a direct action on the fungus at the infection site. Four degradation products plus the parent compound were found in the ambient hydroponic solution and plants after incubating for 72 hr. Three of the degradation products were tentatively identified as 2-(2-hydroxyethylsulfonyl-(acetanilide; 4-phenyl-3-thiomorpholinone 1,1-dioxide; and 2-(vinylsulfonyl)acetanilide. Nutrient solutions in which plants had been grown for three days and were then treated with ^{14}C-oxycarboxin demonstrated degradation products identical to those when plants were left in place. Approximately 50% of the oxycarboxin was degraded. Degradation of oxycarboxin in nutrient solutions was accompanied by increased microbial populations and a rise in pH from 5.6 to 8.0. Plants grown for two weeks in soil containing ^{14}C-oxycarboxin had one degradation product while no degradation of any consequence was seen in the soil. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-7006.)

73-2162. Steen, R. C. (North Dakota State Univ., Fargo, ND). **A study of 2,4-D side-chain degradation in plants.** *Diss. Abstr. Int. 33(9): 4122B; 1973.*

Red-kidney bean (*Phaseolus vulgaris* L.), soybean (*Glycine max* (L.) Merr., var. Clay), pea (*Pisum sativum* L., var. Little Marvel), wild oat (*Avena fatua* L.), barley (*Hordeum vulgare* L., var. Larker), yellow foxtail (*Setaria glauca* (L.) Beauv.), bromegrass (*Bromus inermis* Leyss., var. Manchar Smooth), timothy (*Phleum pratense* L., var Climax), and orchardgrass (*Dactylis glomerata* L., var. Sterling) were treated with 2,4-D by foliar application. 3-(2,4-Dichlorophenoxy)propionic acid [3-(2,4-DP)], a possible intermediate in the side-chain degradation of 2,4-D, was not detected by gas chromatography in any of the plant species during metabolism periods ranging from less than 10 min to 72 hours. In the same plant species, between 0.1% and 3.0% of the absorbed 2,4-D was detected by gas chromatography as 2,4-dichlorophenol five days after hydroponic application of the herbicide. There was no significant difference between monocotyledonous species and dicotyledonous species in the amount of 2,4-dichlorophenol found. 2,4-Dichlorophenol was detected in orchardgrass and bromegrass hydroponically treated with 3-(2,4-DP) in amounts of 2.5% and 9.0% respectively of the 3-(2,4-DP) absorbed in a 24-hour period.

No significant amount of 2,4-dichlorophenol was detected in timothy treated with 3-(2,4-DP) in the same manner. The evidence suggests that 2,4-D is not metabolized to 2,4-dichlorophenol via the 3-(2,4-DP) intermediate. Between 0.2% and 8.0% of the chlorine at position four of the 2,4-D ring was detected by ion-exchange chromatography as inorganic chloride in all nine of the plant species studied. The difference in the chloride produced from 2,4-D between the dicotyledonous species and the monocotyledonous species was not enough to account for resistance of the latter species to the herbicide. Monocotyledonous species contained 9-39% and dicotyledonous species contained 2-5% of the absorbed ^{38}Cl-label from 2,4-D-4-^{36}Cl bound to ethanol-isoluble plant residue. There was no evidence to indicate that inactivation of 2,4-D by conversion to 3-(2,4-DP) accounts for the resistance of monocotyledonous plant species to the herbicide. (Author abstract by permission. Copies of the thesis are available from University Microfilms, Order No. 73-1919.)

73-2163. Mahoney, J. J. (Univ. of Georgia, Athens, GA). **Body storage of DDE and DDT and their effects on migratory physiology and behavior in the white-throated sparrow, *Zonotrichia albicollis*.** *Diss. Abstr. Int. 33(9): 4241B; 1973.*

The onset of spring migratory condition, as measured by *Zugunruhe* and weight increase, was delayed at least one week in White-throated Sparrows, *Zonotrichia albicollis*, fed diets containing 5 or 25 ppm technical DDT. Migratory fat stores, determined by extraction of subsamples sacrified early in the migratory period, were depressed at both treatment levels. However, the difference was significant only at the higher treatment level. Late in ther period there were indications of increased *Zugunruhe* in treated birds. In the high treatment group, *Zugunruhe* is significantly correlated (r = +0.936) with the log of body concentration of DDE which accumulates from the metabolism of ingested DDT. A similar experiment with birds fed DDE (instead of DDT) produced no effect early in the spring migratory period. Late in the period, *Zugunruhe* showed significant dose-dependent increases in treated birds. Daytime activity was not increased significantly. Treatment with DDE in the fall produced no effects on migratory condition. Total body burdens of DDT and its metabolites were significantly higher, at both treatment levels, after 11 weeks at continous treatment than after 5 weeks. For the 5 ppm group the increase was from 115.4 ± 22.0 (x ± se) to 284.7 ± 46.6 micrograms. The gain in the 25 ppm group was from 808.4 ± 45.4 to 2001 ± 232.6 micrograms. Body concentrations reflected this continued accumulation, but increased weight masked a portion of the absolute pesticide gain. At the lower treatment level, the increase in concentration from 18.11 ± 3.41 to 30.72 ± 4.81 ppm, dry weight, was significant. At the higher treatment level the gain from 159.4 ± 10.8 to 217.4 ± 26.1 ppm approached, but did not reach, the significance level. During the fall, residue accumulation was determined after 2,6, and 10 weeks of DDE ingestion. Body concentrations consistently increased significantly at both treatment levels. Birds transferred to clean food for one month after 10 weeks on treatment showed no significant decreases in body or brain residue concentrations. Loss of residues over a 14 week period was determined following DDE treatment for 6 weeks in the spring. Residues were extremely persistent. Half-times for body elimination were calculated to be 130 days for the 5 ppm treatment group and 317 days for the 25 ppm group. Natural depletion of migratory fat stores during the clean food period caused redistribution of some DDE to the brain, resulting in significantly higher brain burdens after a summer on clean food. For birds from the lower treatment level, the increase was from 6.74 ± 0.57 to 21.34 ± 3.09 ppm; for the higher treatment group, from 31.16 ± 4.29 to 72.54 ± 2.38 ppm. (Author abstract by permission. Copies of the thesis are available from University Microfilms, Order No. 73-5732.)

73-2164. Lin, T. H. (Univ. of Kentucky, Lexington, KY). **Influence of selected biologically active chemicals on the mammalian metabolism of carbamate insecticides.** *Diss. Abstr. Int. 33(9): 4318B-4319B; 1973.*

Effects of simultaneous and/or preliminary exposure to carbofuran, ruelene, coumaphos, or DDT and several monoamine oxidase inhibiting drugs were investigated on the metabolism of carbaryl in rats. Radioactive carbaryl given orally as a single dose (50 mg/kg) was completely excreted within 72 hours after treatment, with approximately 80% in the urine and 10% in the feces of the rats. Eleven chloroform-soluble carbaryl metabolites were identified in the urine. Among the water-soluble carbaryl metabolites, 1-naphthyl sulfate was found in relatively large quantities. Seven aglycone metabolites were detected after acid hydrolysis of the water soluble carbaryl metabolites. Simultaneous administration of carbaryl and carbofuran reduced the excretion of carbaryl equivalents by 12%. Ruelene and coumaphos caused a slightly faster excretion of carbaryl equivalents when administered with the carbamate. None of the compounds changed the nature of carbaryl metabolites or changed the amount of carbaryl residues in the body after 168 hours of treatment. Similar results were obtained when these insecticides were administered in the diets of rats. Protein content of the liver, kidney, and spleen were increased by DDT and to a lesser degree by carbaryl. DDT also enhanced mixed function oxidase enzyme activities in the liver and the kidney, whereas carbaryl did not. DDT increased the in vivo glucuronide and sulfate conjugation of 1-naphthol in rats but not the in vitro conjugation of 1-naphthol by rat liver microsomes. Carbaryl did not affect the conjugating enzyme activity in the rat liver. Exposure of rats to the carbamate and/or organophosphates resulted in increased urea and glucose in the urine, reduced the rate of body

weight gains, but had little effect on the pH of the urine. Metabolism and elimination of carbaryl was reduced by treating rats with a series of monoamine oxidase inhibitors. Oxidative and conjugating mechanisms of carbamate metabolism were rendered less efficient by these compounds. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-7355.)

73-2165. Brockway, D. L. (Univ. of Michigan, Ann Arbor, MI). **The uptake, storage, and release of dieldrin and some effects of its release in the fish, *Cichlasoma bimaculatum* (Linnaeus).** *Diss. Abstr. Int. 33(9): 4323B-4324B; 1973.*

Dieldrin uptake, storage, and release from tissues of a tropical fish *Cichlasoma bimaculatum* were investigated. Blood and brain dieldrin concentrations were higher in fish killed by dieldrin than in fish exposed but not killed. After exposure of fish to 1 to 2 ppb of dieldrin for up to 34 days, tissue residue levels had nearly reached a plateau, with whole-body residue concentration factors on a wet-weight basis of 6000 to 11,000. Whole-body concentrations were higher than in brain, and blood concentrations were lowest. After exposure the fish were held in water without dieldrin up to 59 days and subjected to fasting and/or a 5°C rise in temperature showed a very rapid loss of dieldrin and little loss of fat. During the post-exposure period there was no significant increase in blood or brain dieldrin concentration above the final concentration of the exposure phase. The quantity of 17-OHCS metabolites excreted in urine of adult fish was always higher in fish exposed to dieldrin than in controls after the first day of urine collection. The level of hormone excretion decreased continously in exposed fish after the first day. This indicates that there was no increase in stress in the test animals during the period in which dieldrin mobilization from fat was expected. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-6787).

73-2166. Nigg, H. N. (Univ. of Illinois, Urbana-Champaign, IL). **Microsomal enzyme induction by DDT and its analogs in the mouse and chicken.** *Diss. Abstr. Int. 33(10): 4847B; 1973.*

Sixteen DDT-type compounds were tested for their ability to induce hepatic microsomal enyzmes. Biodegradable DDT-type compounds and/or their metabolites were found to be non-inducing in the mouse. Three of the most potent inducers in the mouse were tested on the chicken, but did not induce hepatic microsomal expoxidation, *O*-demethylation or *N*-demethylation. The effect of DDT-type compounds on the microsomal enzyme system was correlated with the physical constants of the compounds. A rational approach for the synthesis of DDT-type compounds which do not induce microsomal enzymes is presented. (Author abstract by permission. Copies of the thesis are available from University Microfilms, Order No. 73-10,014.)

73-2167. Singlevich, T. E. (Marquette Univ., Milwaukee, WI). **Induction of the microsomal drug-metabolizing enzyme system: modification by ethanol and diet.** *Diss. Abstr. Int. 33(10): 4936B-4937B; 1973.*

The effect of chronic administration of ethanol on the activation of the hepatic microsomal drug-metabolizing enzyme system by known inducers such as DDT, chlorcyclizine or 3,4-benzpyrene was investigated. The effect of these agents on hepatic triglyceride levels and the possible modification of the microsomal drug-metabolizing enzyme system by the composition of the diet were also considered. Animals receiving DDT or chlorcyclizine at 50 or 250 ppm, respectively, in a high carbohydrate commercial diet or ethanol as a 15% (v/v) aqueous solution for a period of four weeks showed higher activity of hepatic *O*-demethylase and higher levels of liver cytochrome P-450 than the control animals. When DDT or chlorcyclizine was simultaneously administered along with ethanol, there was a further increase in these two variables. Similar increases in microsomal drug-metabolizing enzyme activity were observed with DDT-ethanol and chlorcyclizine-ethanol, conbinations tested in animals receiving a high fat diet. Ethanol, alone, enhanced the drug-metabolizing enzyme activity in both the high carbohydrate and high fat diets. Furthermore, the liver cytochrome P-450 content of the animals receiving the high fat diet was significantly higher than that of the high carbohydrate group. Regardless of the type of diet, administration of DDT or chlorcyclizine resulted in an increase in hepatic triglyceride levels. This effect was further enhanced by the simultaneous administration of ethanol. However, ethanol given alone did not seem to markedly affect the hepatic triglyceride levels. An investigation into the possible mechanism of effect of inducers on hepatic triglyceride levels has shown that DDT and chlorcyclizine increase the activity of hepatic microsomal palmityl-coenzyme A synthetase, an enzyme which participates in the synthesis of hepatic triglycerides. (Author abstract by permission, abridged. Copies of the thesis are available from University Microfilms, Order No. 73-8301.)

73-2168. Kiriyama, S.; Inoue, S.; Machinaka, N.; Yoshida, A. (Sch. of Nutrition, Fac. of Med., Tokushima Univ., Tokushima, Japan). **[Effects of addition of non-nutritive polysaccharides on toxicity of dietary polychlorinated biphenyls (PCB) and 1,1,1-trichloro-2, 2-bis(*p*-chlorophenyl) ethane(*p,p'*-DDT).]** *Eiyo To Shokuryo (J. Jap. Soc. Food Nutr.) 26(1): 15-25; 1973.* (24 references) (Japanese)

The growth rates, various organ weights, and plasma and liver cholesterol were examined in male weanling Wistar rats fed diets containing various levels of *p,p'*DDT (0.002, 0.02 and 0.2%). The effects of addition

of konjac mannan, pectin, carboxymethyl cellulose, anion exchange resin (AG 2-X8, Cl form), or sodium cholate on these indices were also studied. Plasma cholesterol levels were significantly increased in rats fed 0.2% DDT but not at the lower DDT levels. Addition of konjac mannan to the diet containing 0.02% DDT caused significantly low plasma cholesterol levels as compared with the value in rats fed 0.02% DDT diet. The 0.2% DDT level caused whole body convulsions although the animals continued to grow but at a slower rate.

73-2169. Armbrecht, B. H.; Shalkop, W. T. (FDA, HEW, Agr. Res. Cent., Beltsville, MD 20705). **The effects of DDT and aflatoxin priming on the acute aflatoxin toxicity in rams.** *Environ. Physiol. Biochem. 2(1): 3-6; 1972.* (14 references)

No death occurred among sheep pretreated with 80 μg of aflatoxin per kg or 50 mg *o,p'*-DDT per kg for five days and challenged after two days rest with 2 mg of aflatoxin B_1 per kg. The 1 day LD50 for aflatoxin in rams primed for 10 days with DDT was 2.8 mg/kg. During the priming with DDT, no toxic signs were observed and animals gained 1.5 to 3 kg/wk. Hepatic response, the same in primed and unprimed animals that died within a day, included marked centrilobar hemorrhagic necrosis with few normal liver cells remaining in the portal and septal areas. In primed animals exposed to 4 mg/kg aflatoxin histopathologic changes included marked fatty vacuolation of liver cells and an early bile duct proliferation reaction. These were not evident in the nonprimed sheep.

73-2170. Phillips, W. E. J.; Hatina, G.; Villeneuve, D. C.; Grant, D. L. (Res. Lab., Health Protect. Branch, Dept. of Nat. Health and Welfare, Ottawa, Canada). **Effect of parathion administration in rats following long term feeding with PCB's.** *Environ. Physiol. Biochem. 2(4): 165-169; 1972.* (11 references)

Polychlorinated biphenyls administered in the diet for one year did not potentiate parathion toxicity in the rat. Plasma ChE was decreased in animals fed Aroclor 1254 at 200 ppm. A single dose of parathion depressed plasma ChE to approximately the same extent in controls and rats pretreated with Aroclor 1221 and 1254. Brain ChE activity was increased in animals fed Aroclor 1221 at 220 ppm, but no parathion-Aroclor interactions were observed in connection with brain ChE. Aroclor 1221 at concentrations up to 200 ppm had no significant effect on body weight. Increased liver weight and decreased body weight were observed with Aroclor 1254 at 200 ppm.

73-2171. Adams, G. K.; Yamamura, H. I.; O'Leary, J. F. (College Pharm., Univ. Kentucky, Lexington, KY 40500). **Spontaneous recovery of central respiratory function after an irreversible AChE inhibitor.** *Eur. J. Pharmacol. 20(3): 377-380; 1972.* (8 references)

Intravenous administration of Soman at 38 mg/kg to guinea pigs consistently blocked complete respiration as monitored by tracheal airflow and phrenic nerve activity. The effect on blood pressure was variable, and the compound produced a severe persistent depression of brainstem AChE. Adequate ventilatory flow was restored spontaneously within an hour after administration of the compound. Although a second dose of 19 mg/kg of Soman further depressed brainstem AChE levels, central respiratory function remained stable. When multiple doses of Soman were administered at various times after spontaneous return of respiratory function, no central respiratory paralysis was observed although brainstem AChE levels were reduced to nonmeasurable levels. The data suggests that recovery of central respiratory function after Soman intoxication is not a result of a return of brainstem AChE activity.

73-2172. Singlevich, T. E.; Barboriak, J. J.; Pintar, K. (Dept. Pharmacol. Pathol., Med. Coll. Wisconsin, Milwaukee, WI). **Modification by ethanol of the hepatic triglyceride response to chronic administration of chlorcyclizine and DDT.** *Eur. J. Toxicol. 4(6): 532-535; 1971.* (9 references)

Chlorcyclizine or DDT, both microsomal enzyme inducers, were incorporated in diets at concentrations of 0.025% and 0.005%, respectively, and fed for 28 day periods to male albino Sprague-Dawley rats to study the effect on hepatic triglyceride response to chronic ethanol administration. DDT-treated animals had a hepatic triglyceride concentration of 8.69 mg/g of liver; the chlorcyclizine group, 13.6 mg/g; and the control group, 6.1 mg/g. Ethanol did not affect hepatic triglycerides. The chlorcyclizine and DDT-induced increases in liver triglyceride content was enhanced by simultaneous administration of ethanol. Histological examination showed again that chlorcyclizine or DDT, but not ethanol, caused fatty infiltration only of the chlorcyclizine group.

73-2173. Baluja, G.; Murado, M. A. (Inst. Gen Org. Chem., Madrid 6, Spain). **Metabolism of aldrin by *Penicillium glaucum*.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 273-281.* (8 references)

Aldrin oxidation yielded dieldrin and two other metabolites in an isolated species of *Penicillium glaucum* cultured in liquid and solid media with doses of aldrin ranging from 0.08 to 1.6 $\mu g/cm^2$. These two additional metabolites were of slightly higher polarity than dieldrin. The failure of the microorganism to produce an ultimate degradation of dieldrin suggests two metabolic pathways are involved in the *P. glaucum* conversion of aldrin. One pathway leads directly to dieldrin by epoxidation. The other leads to the two other metabolites by direct conversion of aldrin. The metabolic pathway is affected by the concentration of insecticide, higher con-

centrations of aldrin yielding dieldrin as the metabolite of preference.

73-2174. Gorbach, S. (Farbwerke Hoechst AG., Frankfurt, Germany). **Terminal residues of endosulfan.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 283-286.* (4 references)

In mammals endosulfan is metabolized to endosulfan-sulfate, endosulfan-alcohol, endosulfan-hydroxyether, and endosulfan-lactone. Endosulfan itself was detected only in the feces, the sulfate in fatty tissues and milk, the hydroxyether and lactone in the urine. The sulfate carries the same toxicity for mammals as endosulfan itself, whereas the diol, hydroxyether, and lactone may be considered nontoxic. Sulfate residues in fatty tissues were below 1 ppm and were detoxified and excreted within 14 days after endosulfan treatment was stopped. Endosulfan was not persistent even in fish. Algae showed no intoxication although they absorbed the insecticide within a few hours after exposure to a concentration of 10 ppm. Bacteria from sewage mud gave similar results. The toxicological relevance of terminal residues in the environment was studied in a large scale situation. Immediately after application in the rainy season the average river residue was 0.4 ppb. Sea water and usually fish ponds as well were entirely free from residues.

73-2175. Casida, J. E. (Div. Entomol., Univ. California, Berkeley, CA 94720). **Chemistry and metabolism of terminal residues of organophosphorus compounds and carbamates.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 295-314.*

Studies on pesticide metabolism have increased in recent years. Advances in technology have permitted closer and more thorough examinations of metabolites. The addition of new pesticides to the field is diminishing, allowing time to catch up on those already on the market. It is now possible to define the stages of metabolism through which a pesticide passes until it reaches a terminal residue. This is frequently done in rats and in the crop plant which will be treated with the chemical as well as a host of additional organisms and plants which may contact the pesticide. Fortunately, metabolic systems are similar enough in most organisms so that a few critical representative examples can be applied to other systems not able to be directly studied. The metabolic pathways which are deemed of toxicological significance must be the ones included in residue considerations. Most of the metabolites ultimately enter normal metabolic pools. Usually, fragments which have entered these pools persist for a long time, but are unable to be released by subsequent organisms in the food chain. Thus the science of pesticide metabolism has developed to such an extent that it can now be used in predicting the occurrence of hazardous residues.

73-2176. Spencer, E. Y. (Res. Inst., Canada Dept. Agr., London, Ontario). **Chemistry and metabolism of terminal residues of organophosphorus compounds and carbamates.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, 315-318.*

The following points are highlighted in this comment concerning a more detailed report on pesticide metabolism. The *N*-oxide derivative of schradan may well exist as a transitory intermediate during the metabolic process, but there is no direct evidence for it. The active toxic intermediate formed by dehydrochlorination and rearrangement of trichlorfon is apparently dichlorvos. Further study is needed on reported inhibition of carboxyesterase and resulting increases in mammalian toxicity of malathion during administration of frequent, normally sublethal, doses. Work is expanding on the specificity of transmethylation enzymes to other dimethyl vinyl phosphates such as phosphamidon. A more intensive study of metabolism and residues in the soil is advised.

73-2177. Miyamoto, J. (Inst. Phys. Chem. Res., Saitama, Japan). **Metabolism of organophosphorus compounds and carbamate insecticides.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 319-324.* (19 references)

Trichlorfon has been non-enzymatically converted to dichlorvos. Dichlorvos, not trichlorfon, is the biologically active form. Fenitrothion has remarkably low toxicity to mammals. This, plus its high efficacy against insects, makes it extremely useful. Conversion of fenitrothion to its toxic oxygen analog, fenitrooxon, was hardly detectable in oral administration, but was demonstrated in intravenous administration. This conversion is catalyzed by NADPH-dependent microsomal enzymes of mammalian liver. Orally administered fenitrothion-phosphorus-32 was 90% recovered in the urine and 10% in the feces during a four day period in guinea pigs. Forty-six days after application of phosphorus-32 labeled fenitrothion in rice plants, 0.7 ppb of fenitrothion was present in the plants. Some fenitrooxon was detected, but it disappeared rapidly. Under aerobic conditions at 37°C, *Bacillus subtilis* reduced 65% of phosphorus-32 fenitrothion in 48 hours. Fenitrooxon was not detected. Feeding 100 mg/kg/day of fenitrothion to cattle caused no adverse effects. Cyanox was absorbed from the gastrointestinal tract of male rats and distributed to various tissues. Some Cyanoxon was formed. More than 90% of the chemical passed in the urine in 96 hours. Biodegradation of Cyanox and Cyanoxon in mammals occurs at the *O*-alkyl and *O*-aryl bonds; the *p*-cyano group is not attacked. Meobal biodegradation was studied in rats. The 4-hydroxymethyl analog of Meobal inhibited mammalian cholinesterase and had efficacy to insects and toxicity to mammals nearly equivalent to that of Meobal. Meobal biodegradation proceeds through oxidative pathways. Meobal applied at the rate of 3 kg/0.1 ha over rice was detected in the grains

two weeks later at 0.11 ppm. In 30 to 50 days this decreased to 0.014 ppm.

73-2178. Slade, P. (ICI Plant Protection Ltd., Jealott's Hill Res Sta., Berks, England). **Metabolism of carbamate and thiocarbamate herbicides.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 325-331.* (25 references)

Phenyl-carbamate herbicides do not generally hydrolyze in plants although hydrolysis may be important in resistant plants. Detoxification usually arises by hydroxylation and conjugation with plant constituents. Microbial degradation accounts for the disappearance of propham from soils. Other phenylcarbamate herbicides degrade similarly but leave some persistent substituted anilines. Rats with intravenously administered propham excreted the *O*-sulfate of the product of *p*-hydroxylation of the herbicide. The rate of degradation of 3,4-dichlorobenzyl-*N*-methylcarbamate (UC 22463) depends on the sensitivity of the plant. Metabolites in plants occur as water soluble conjugates hydrolysed by β-glucosidase. In the rat metabolites are conjugated as glucuronides or a 3,4-dichlorohippuric acid before excretion. Many metabolites have been observed for the thiocarbamate and dithiocarbamate herbicides EPTC, pebulate, vernolate, and diallate, but they have not been identified. A synergistic effect occurs when methylcarbamate insecticides such as carbaryl strongly inhibit the enzymic hydrolysis of chlorpropham, thus increasing the persistence of this herbicide.

73-2179. Nachtomi, E.; Alumot, E.; Bondi, A. (Dept. Animal Nutr., Volcani Inst. Agr. Res., Rehovot, Israel). **Metabolism of halogen containing fumigants in mammalians and birds.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 495-501.* (21 references)

Biochemical reactions caused by ethylene dibromide, carbon tetrachloride, and other fumigants were studied in rats and chicks. *N*-acetyl-*S*-(β-hydroxyethyl)-cysteine was the main metabolite found in the urine of ethylene dibromide exposed rats; S(β-hydroxyethyl)-cysteine was the second metabolite. When ethylene dichloride or ethylene bromohydrin were administered to rats, the same metabolites were detected. The degradation of S-(β-hydroxyethyl)glutathione to cysteine derivatives and the final acetylation to mercapturic acid occurred mainly in the kidneys. When carbon tetrachloride was given to rats, no sulfur containing detoxification compounds were detected in the urine. However, a striking decrease in thiol groups was found in livers of animals treated with both ethylene dibromide and carbon tetrachloride. A similar drop of liver sulfhydryl groups was found in chicks treated with both substances. No increased in liver fat levels was found in chicks after carbon tetrachloride treatment. The fact that sulfhydryl groups and ascorbic acid levels dropped during poisoning with ethylene dibromide and carbon tetrachloride in both species suggests a free radical reaction. In these experiments the diene conjugation observed in the microsomal lipid of ethylene dibromide poisoned rats attained only half the level in carbon tetrachloride treated animals. Liver fat accumulation was much more pronounced in carbon tetrachloride than in ethylene dibromide poisoning. The metabolic rate of ethylene dibromide was more pronounced than that of carbon tetrachloride in the livers of rats. It is concluded that ethylene dibromide exerts its toxic effect by changing the oxidation-reduction equilibrium, due to the detoxification mechanisms, while carbon tetrachloride exerts its effect by its prooxidative action on polyunsaturated lipids.

73-2180. Fishbein, L.; Falk, H. L.; Fawkes, J.; Jordan, S. (Nat. Inst. Environ. Health Sci., Research Triangle Park, NC). **The metabolism of ^{14}C-piperonyl butoxide in the rat.** *In: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 503-519.* (16 references)

The metabolism of orally and intratracheally administered labeled piperonyl butoxide was studied by paper, thin-layer radioautographic, and liquid-scintillation techniques. Results were compared to earlier studies using the intravenous route of administration. The large numbers of metabolites found in the bile, urine, and feces on all three routes of entry demonstrate that many sites on piperonyl butoxide are subject to attack. With intravenous administration there is widespread tissue distribution of the chemical along with prolonged biliary and urinary elimination of many metabolites. After oral administration the synergist, with or without Wesson oil, undergoes rather poor absorption from the gastrointestinal tract as well as rapid elimination in the urine and feces. Intratracheal administration results in a prolonged period of elimination in both bile and urine subsequent to a high initial level of biliary excretion. Both oral and intratracheal administration resulted in lower lung tissue residues of unmetabolized synergist than that following intravenous administration. These studies, coupled with an understanding of the stability of the synergist, illuminate certain hazards in repeated and prolonged contact or in inhalation of methylenedioxyphenyl synergists.

73-2181. Wassermann, M.; Wassermann, D. (Hebrew Univ.-Hadassah Med. Sch., Jerusalem, Israel). **Effects of organochlorine insecticides on homeostatic and immunologic processes.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 521-529.* (9 references)

In a study on two groups of workers, one occupationally and the other non-occupationally exposed to organochlorine insecticides, the serum cholesterol level was significantly higher in occupationally exposed

workers only in the 45 yr and older group. In this age group the homeostatic control of serum thyroxine is impaired and the breakdown of cholesterol may be diminished, thus the increased cholesterol synthesis induced by organochlorine insecticides is no longer masked by a concomitant catabolism. The serum PBI was considerably lower in occupationally exposed workers. It is suggested that the organochlorine insecticides in the body change the ratio of free thyroxine. To test the influence of these chemicals in the area of immunological response to a soluble or particulate antigen, rats were given 200 ppm DDT in the drinking water for 35 days and a total of 6 mg ovalbumin in three weekly injections. A 30% decrease of antiovalbumin antibodies resulted. Rats receiving 200 ppm DDT only showed a rise of albumin and lowering of gamma globulin fractions of serum proteins. Rabbits receiving 200 ppm DDT had impaired immunological responses. The total gamma globulins were significantly reduced in DDT-Salmonella treated rabbits, but in those receiving sheep red blood cell antigen, the antibody titer decrease was not significant. The difference in impairment with antigens of *Salmonella typhi* vs SRBC may arise because the DDT plasma level was higher in Salmonella than SRBC rabbits. This suggests that the plasma level of total DDT depends on the nature of the antigen and that the immunological response is inversely proportional to the amount of total DDT in the plasma. These conclusions are vital when implications in drug detoxication and immunological response are considered.

73-2182. Brown, J. R. (Univ. Toronto, Toronto, Canada). **The effect of dietary Kelthane on mouse and rat reproduction.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, 531-548.* (7 references)

Five generations of rats and mice were fed Kelthane (dicofol) in their food in concentrations of 0, 7, 25, 100, 225, and 500 ppm. A marked difference was noted between rats and mice; no offspring were produced in rats receiving more than 100 ppm Kelthane in their diet. In mice receiving doses up to 225 ppm no differences were observed with respect to litter size at birth and at 21 days nor with the individual weights of siblings at 21 days. In the 500 ppm group litter size at birth and at 21 days, as well as individual weights of animals, were lowered. In groups through the 225 ppm level no differences existed in fertility, viability, or lactation indices whereas these values were all lowered in the 500 ppm dose range. No fetal abnormalities were detected in any of the generations even in the mice at the 500 ppm dietary level. Pathological examinations of the tissues of animals of the fifth generation showed no significant difference from the group receiving no dietary Kelthane.

73-2183. Street, J. C.; Blau, S. E. (Animal Sci. Dept., Utah State Univ., Logan, Utah). **Oxychlordane: accumulation in rat adipose tissue on feeding chlordane isomers or technical chlordane.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, p. 549.*

Male and female rats were fed pure cis- and/or trans-chlordane or technical chlordane at 25 to 100 ppm for 15 days. Residues of oxychlordane and the parent compounds found in the adipose tissues were analyzed by EC-GLC. Trans-chlordane is more reactive, allowing greater oxychlordane storage and lower parent residues for both sexes. The storage ratios at 100 ppm of oxychlordane:chlordane were 20.0 and 9.7 (female and male) for the trans-isomer, but 4.3 and 3.4 for the cis. At all isomeric proportions the oxychlordane formation is simply additive. There is no apparent interaction. Oxychlordane storage from technical chlordane can therefore be predicted from the isomeric content of the technical mixture.

73-2184. Kuz'minskaya, U. A.; Novachik, V.; Klisenko, M. A. (All-Union Sci. Res. Inst. Hyg. Toxicol. Pestic., Polymers and Plastic Materials, Kiev, USSR). **Raspredeleniye polikhlorkamfena v lipidnykh fraktsiyakh tkaney. [Distribution of polychlorocamphene in lipid fractions of tissues.]** *Gig. Sanit. 37(12): 96-97; 1972.* (2 references) (Russian)

The distribution of polychlorocamphene (toxaphene) was studied by thin-layer chromatography in lipid fractions of liver, brain, and medulla oblongata tissues of albino rats fed single 120 and 24 mg/kg, or daily 2.4 mg/kg doses of polychlorocamphene for 3-6 months. Liver tissues contained polychlorocamphene only in free form, not associated with any lipid fraction. After administration of a single 24 mg/kg dose, the hepatic residue content decreased to 1.1 mg/kg from 2.25 mg/kg between the 1st and the 5th day; residues disappeared completely in 15 days. A close relationship between the hepatic residue content and dose was established. The residue level was independent of the duration of exposure in the chronic feeding test. Polychlorocamphene appeared in lipid fractions of the brain and medulla oblongata 15 days after intake of a single 120 mg/kg dose. The residue levels in phospholipids, free cholesterol, triglycerides, and cholesterol esters were 0.9 μg/g, 0.2 μg/g, and 1.08 μg/g. The polychlorocamphene level in the brain phospholipid fraction increased by 66% as the administration of daily 2.4 mg/kg doses was extended from 3 to 6 months. The corresponding increase in the polychlorocamphene residue levels in phospholipids, free cholesterol, and another unidentified lipid fraction of the medulla oblongata averaged 65%.

73-2185. Nakamura, A.; Hara, T.; Minakami, S. (Dept. Biochem., Kyushu Univ. Sch. Med., Fukuoka, Japan). **Intracellular distribution of hepatic catalase activity in rats treated with aminotriazole and allylisopropylacetamide.** *J. Biochem. 73(1): 47-53; 1973.* (23 references)

Changes in the intracellular distribution of catalase activity were studied in male Wistar rats given amitrole (100 mg/100 g i.p.) and allylisopropylacetamide (20 mg/100 g i.p. every 12 hr). Ninety minutes after amitrole injection, ethanol (100 mg/100 g) was injected to completely suppress the inhibitory effect of amitrole. The hepatic catalase activity decreased rapidly after injection of amitrole reaching a minimum of 10% normal value in 1.5 hr. At this time, the activity in microsomes was 20% of the normal value whereas activity in peroxisomes and the cytosol was less than 10%, probably due to the continuous synthesis of the enzyme in the presence of amitrole. The activity began to increase about 3 hr after amitrole injection and returned to the original level more rapidly in the peroxisomes than in the microsomes. This suggests that the newly synthesized catalase is immediately and preferentially transferred to peroxisomes. Allylisopropylacetamide gradually decreased the hepatic catalase activity, and after the first 2 days the activity was 25% of that of controls. Twelve hours after the first injection microsomal and cytosol activities were decreased 50%. The activity in peroxisomes did not start to fall until the activity in the microsomal fraction had decreased to a low level. This supports the idea that the catalase is synthesized in microsomes and then transferred to peroxisomes. The change in catalase activity in the cytosol differed from that in peroxisomes both during recovery after amitrole treatment and during degradation after allylisopropylacetamide treatment.

73-2186. Atwal, O. S. (Anatom. Lab., Dept. Biomed. Sci., Ontario Vet. College, Univ. Guelph, Guelph, Ontario, Canada). **Fatty changes and hepatic cell excretion in avian liver. An electron microscopical study of Kepone toxicity.** *J. Comp. Pathol. 83(1): 115-124; 1973.* (17 references)

Intramuscular injection of 0.5 mg of Kepone (chlordecone) daily for 10 days in Japanese quail resulted in several ultrastructural changes in the liver. The early change was disappearance of glycogen. Mitochondria appeared to be enlarged and swollen, and rough endoplasmic reticulum was moderately dilated. Hepatic cells developed large lipid vacuoles and mitochondria grouped around the lipid vacuoles. Autophagic vacuoles developed, particularly in the pericanalicular side, and were extruded into the bile canaliculi and into the vascular channels. The changes underlying the excretion of cellular debris into the biliary canaliculi and vascular channels may signify a morphological basis of rapid loss of Kepone residues from the liver cells.

73-2187. Desmarchelier, J.; Krieger, R. I.; Lee, P. W.; Fukuto, T. R. (Div. of Entomol., C.S.I.R.O., P.O. Box 109, Canberra, A. C. T., Australia). **Carbaryl synergism by substituted 1,3-benzodioxoles and related compounds and inhibition of mixed function oxidase.** *J. Econ. Entomol. 66(3): 631-638; 1973.* (32 references)

Several new 1,3-benzodioxole derivatives were synthesized and examined for synergism of carbaryl toxicity to houseflies, *Musca domestica* L., and for inhibition of aldrin epoxidation by rat liver microsomal oxidase. Synergism data for several of the derivatives confirmed predictions made earlier by another worker. Several of the 4,5-disubstituted-1,3-benzodioxoles were outstanding in their effectiveness in synergizing the toxicity of carbaryl. A direct relationship between synergistic activity and inhibition of microsomal aldrin epoxidation was not observed. Data on inhibition of microsomal oxidase revealed that the catechols are often more potent inhibitors than the related 1,3-benzodioxoles.

73-2188. Mann, H. D.; Ivey, M. C.; Kunz, S. E.; Hogan, B. F. (U. S. Livestock Insects Lab., Agr. Res. Serv., U. S. Dept. of Agr., Kerrville, TX 78028). **Chlorpyrifos, its oxygen analogue, and 3,5,6-trichloro-2-pyridinol: residues in the body tissues of turkeys confined in pens on treated soil.** *J. Econ. Entomol. 66(3): 715-717; 1973.* (3 references)

Turkeys were confined in a pen on soil that was sprayed with chlorpyrifos wettable powder at four pounds active ingredient per acre (4.48 kg per hectare). A portion of the soil was treated again 28 days later with the same dosage. Residues in fat, skin, muscle, liver, and kidney were determined for chlorpyrifos and 3,5,6-trichloro-2-pyridinol, a likely metabolite; and in fat, skin, and muscle for the oxygen analog (diethyl 3,5,6-trichloro-2-pyridyl phosphate). Maximum residues of chlorpyrifos (0.055 ppm in fat, 0.152 ppm in skin, and 0.005 ppm in kidney) were detected seven days after the first application; no oxygen analog was detected. Residues of 0.003-0.165 ppm pyridinol were found in skin, liver, and kidney tissues of some turkeys seven days after both the 1st and the 2nd applications. No increase in the residues of either material above the levels found seven days after the first treatment resulted from the second treatment. (Author abstract by permission)

73-2189. Johnson, G. A.; Jalal, S. M. (Biol. Dept., Univ. N. Dakota, Grand Forks, ND 58201). **DDT-induced chromosomal damage in mice.** *J. Heredity 64(1): 7-8; 1973.* (10 references)

Mice treated with 100 to 400 mg/kg DDT had significantly higher proportions of chromosomal aberrations, which were studied in the bone marrow, than controls. Deletions in the form of fragments, one of the most common forms of abnormality encountered, occurred at an average frequency of 0.41% in controls and at 3.0, 1.5, 10.0, and 9.2% in mice treated with 100, 150, 200, and 300 to 400 mg/kg, respectively. Stickiness and occasional ring and metacentric chromosomes were the other abnormalities observed. DDT appears to be a potential mutagen, as has been suggested by other investigations.

73-2190. Shull, L. R.; Cheeke, P. R. (Dept. Animal Sci., Oregon State Univ., Corvallis, OR). **Antiselenium activity of tri-*o*-cresyl phosphate in rats and Japanese quail.** *J. Nutr. 103(4): 560-568; 1973.* (49 references)

Tri-ortho-cresyl phosphate (TOCP) added to low selenium-low vitamin E Torula yeast based diets resulted in growth cessation and mortality in both rats and Japanese quail. In growing rats supplementation of the diets with various levels of either selenium or vitamin E prevented both growth cessation and mortality. Rats that had ceased growing after TOCP administration responded with renewed growth when the diet was supplemented with either vitamin E or selenium. The response to selenium was significantly greater than the response to vitamin E. There was a slight tendency for TOCP to increase the specific activity of lipase in pancreatic homogenates. With TOCP-fed Japanese quail, the gains were significantly greater and the mortality lower when selenium was supplemented than when vitamin E was added. The results in both rats and Japanese quail suggest that TOCP antagonizes selenium as well as vitamin E. The formation of one or more compounds of Se and a metabolite of TOCP is suggested to account for a TOCP-Se antagonism. The possibilities include a selenophosphorus excretory compound, an excretory compound with selenium bound to *o*-cresol, and an excretory compound with selenium in combination with ethereal sulfate and the neutral sulfur fraction of urine.

73-2191. Carter, F. L.; Graves, J. B. (Dept. Entomol., Univ. Arkansas, Fayetteville, AR 72701). **Measuring effects of insecticides on aquatic animals.** *La. Agr. 16(2): 14-15; 1973.*

Insecticides selected on the basis of their present or potential value in pest control programs were tested for toxicity to white river crawfish, three species of fish, and bullfrog tadpoles. Crawfish were most sensitive to the pesticides and bullfrog tadpoles the least sensitive. Methyl parathion was 2,033 times more toxic to crawfish than to the tadpoles when LC50s were compared. Bluegill was the most sensitive of the fish tested. Insecticides used were chlorpyrifos, methyl parathion, azinphosmethyl, monocrotophos, dicrotophos, malathion, carbaryl, carbofuran, methomyl, aldicarb, DDT, toxaphene, toxaphene plus DDT, and mirex. Generally, the organochlorines were the most toxic group of insecticides and carbamates the least toxic. Two of the organophosphates, malathion and dicrotophos, were only slightly toxic to all of the test species. Clearly, higher animals are less sensitive to insecticides than are lower forms, and responses vary according to species.

73-2192. Epifanio, C. E. (Coll. Mar. Studies, Field Sta., Univ. Delaware, Lewes, DE 19958). **Dieldrin uptake by larvae of the crab *Leptodius floridanus*.** *Mar. Biol. 19(4): 320-322; 1973.* (10 references)

Leptodius floridanus larvae accumulated dieldrin 19.1 times as fast from 0.5 ppb in seawater as from 213 ppb in food. Calculations based on their pumping rates and on feeding rates estimated from those of the stone crab indicate that larvae reared in seawater containing 0.5 ppb dieldrin are exposed to at least 1.55 times as much pesticide as those fed *Artemia salina* nauplii contaminated with 213 ppb dieldrin. If a straight line relationship exists between dose factors and rate of uptake factors and the slope is 1.0, then *Leptodius* larvae would accumulate dieldrin 8128 times as fast from the water as from the food having the same concentration. Under field conditions the larvae would accumulate dieldrin 1.23 times as fast from the water as from the food, suggesting that both routes are important in the accumulation of organochlorines by small carnivorous zooplankters.

73-2193. Faff, J. (Dept. of Exp. Pharmacol., Med. Acad., Warsaw, Poland). **Badania nad toksycznoscia estru pinakolinowego kwasu metylofluorofosfonowego dla szczurow w okresie ich zdrowienia po ostrym zatruciu tym inhibitorem. [Toxicity of pinacolyl methylphosphonofluoridate to rats during the recovery period following severe poisoning with this inhibitor.]** *Med. Pr. 23(2) 121-128; 1972.* (28 references) (Polish)

Increased sensitivity of rats to soman after acute poisoning was studied. The LD50 and acetylcholinesterase (AChE) were determined at different intervals in surviving rats after intramuscular injection of either 50 μg/kg (group I) or 80 μg/kg followed immediately by 20 mg/kg atropine (group II). In comparison to controls, (LD50=66 μg/kg) the LD50 in group I was reduced to 42.4 μg/kg (64% of original) and in group II to 33.6 μg/kg (51% respectively) after one day. The sensitivity to a repeated injection was elevated up to 6 days in group I and 14 days in group II. AChE in brain, erythrocytes, and serum was more markedly reduced in group II than group I, and returned quicker to near normal levels in group I than group II (50% activity of brain AChE 48 hours after intoxication in group I as compared to 6-14 days in group II respectively.) It is concluded that increased sensitivity to soman, which persists for several days after disappearance of visible symptoms of intoxication, is related to low levels of AChE, but normal reaction returns earlier than full regeneration of AChE in brain or erythrocytes.

73-2194. Ashton, F. M.; Crafts, A. S. (Dept. Botany, Univ. California, Davis, CA). **Molecular fate of herbicides in higher plants.** *IN: Mode of Action of Herbicides, John Wiley & Sons, New York, 1973, pp. 62-68.* (7 references)

Representative examples illustrate the types of reactions involved in herbicide degradation in higher plants and demonstrate the formation of inactive and more active products in the environment. The phenoxy herbicides undergo three different types of oxidation

involving three different sites on the side chain. Phenoxy, benzoic, and urea derivatives undergo decarboxylation. Hydroxylation has been demonstrated with derivatives of the phenoxy, benzoic, and triazine classes. In the latter class ring hydroxylation involves dechlorination, demethoxylation, or demethylthioation. The hydrolysis of a carbamate molecule illustrates the major split which is characteristic of this reaction and which generally leads to two relatively large non-phytotoxic fragments. Dealkylation has resulted in varied selectivity to different plants, particularly in the case of the triazine and urea type herbicides. Conjugation of herbicides has been reported frequently with sugars and amino acids and sometimes with protein or lignin. Ring cleavage appears to occur slowly, if at all, in higher plants.

73-2195. Parry, J. M. (Dept. Genetics, Univ. College of Swansea, Singleton Park, Swansea SA2 8PP, Wales). **The induction of gene conversion in yeast by herbicide preparations.** *Mutat. Res. 21(2): 83-91; 1973.* (23 references)

The technique of prototrophic induction produced by gene conversion in the yeast *Saccharomyces cerevisiae* was utilized to test the genetic activity of three herbicide preparations containing paraquat, mecoprop (MCPP) and dinoseb in an attempt to standardize the screening of such herbicides. Both paraquat and dinoseb induced conversion of genes at the ade-2 and his-1 loci at high survival levels, while mecoprop induced conversion only at low survival levels. With each herbicide the peak range of activity occurred at concentrations used for spray purposes. Although the implications of these findings in terms of human genetic changes cannot be accurately assessed, the problems of virulence and resistance of plant pathogens which could be induced by such herbicides are evident.

73-2196. Dassenoy, B.; Meyer, J. A. (Univ. Catholique de Louvain, Lab. Phytopathol. Mycol. Generale, 42 de Croylaan, Heverlee, Belgium). **Mutagenic effect of benomyl on *Fusarium oxysporum*.** *Mutat. Res. 21(2): 119-120; 1973.* (7 references)

Benomyl, at 5 μg/ml, which is less than spray concentration, induced forward mutations in growing cells of *Fusarium oxysporum* f. sp. *melonis*, according to a short communication. In the back mutation test performed with three monoauxotrophs no growth was observed with the control and with benomyl-treated conidia. No chromosomal aberrations were observed in roots of young *Allium cepa* treated with benomyl or in control roots. The results call attention to the necessity for testing pesticides for mutagenic activity on various organisms and under different conditions.

73-2197. Nagasaki, H. (Sch. of Hyg., Nara Prefect. Med. Coll., Japan). **[Experimental studies on chronic toxicity of benzene hexachloride (BHC).]** *Nara Igaku Zasshi (J. Nara Med. Ass.) 24(1,2): 1-26; 1973.* (38 references) (Japanese)

Studies were carried out on the chronic toxicity and carcinogenicity of technical, α,β,γ,and δ-BHC given to male dd-mice in a standard daily diet. Pathological findings were studied in brain, heart, liver, kidney, spleen, testes, lungs, thyroid, adrenals, bladder, and bone marrow of mice treated with each compound. Residual levels of BHC were measured in liver, brain, kidney, testes, and adipose tissue. The mice were given technical BHC respectively at the level of 660.0, 66.0, and 6.6 ppm of basal diet for 24 weeks. Those fed 660.0 ppm developed many liver tumors which had clear signs of malignancy. Hepato-carcinogenicity was investigated by administering α,β,γ and δ-isomer, respectively, at the levels of 500, 250, and 100 ppm of the standard diet for 24 weeks. All mice (20) treated with 500 ppm of α-isomer and some (17/20) of mice treated with 250 ppm of α-isomer developed liver tumors with signs of malignancy. No remarkable changes were observed on other groups of mice. After administering technical BHC for 24 weeks, the residual ratios and amounts of the four isomers differed from the ratio in the administered BHC. The residual levels in organs increased in proportion to the administered amount of each isomer. High levels of α-and β-isomer were seen, especially in adipose tissues and liver. It was concluded that α-isomer of BHC induces hepato-carcinogenicity in male dd-mouse.

73-2198. Vohland, H. W.; Koransky, W.; Zufelde, H. (Inst. fuer Toxikol. und Pharmakol. der Philipps-Univ. Marburg, Germany). **Effect of α-hexachlorocyclohexane on the convulsive activity of pentetrazol (Cardiazol) in the rat.** *Naunyn-Schmiedebergs Arch. Pharmakol. 275(3): 289-298; 1972.* (24 references)

A single dose of α-hexachlorocyclohexane (α-BHC) induces tolerance to the convulsive action of pentetrazol. The increase in pentetrazol seizure threshold becomes significant 3 hr after administration of 200 mg/kg α-BHC and reaches a maximum 12-24 hr after pretreatment. The effect is shown to persist for about 9 days. Pentetrazol concentrations in brain and other organs during the maximum of anticonvulsive activity of μ-BHC were unaffected in α-BHC pretreated animals. There is a linear relationship between α-BHC content of the brain and the concentration of pentetrazol when generalized convulsions occur. Increase in α-BHC content of the brain is accompanied by a decrease in the sensitivity of the central nervous system for pentetrazol. (Author abstract by permission)

73-2199. Nooden, L. D. (Botany Dept., Univ. Michigan, Ann Arbor, MI 48104). **Maleic hydrazide: another plant growth regulator threatens human health.** *Newslett. Environ. Mutagen Soc. 3: 9-10; 1970.* (10 references)

A number of widely used herbicides, probably

more harmful than those being examined carefully, are going unnoticed in the public campaign to eliminate highly toxic materials. Maleic hydrazide, a synthetic plant-growth regulator and herbicide, inhibited cell division and disrupted normal development in plants as early as 1949. Recently, investigators tested the compound in animals and found mutagenic, carcinogenic, and antimitotic effects. In plants the amount of maleic hydrazide required to break chromosomes is well below the amount required to inhibit cell division, and it is probable that similar levels produce similar effects in plants and animals. Because of this it is surprising that the use of MH is expanding, rather than decreasing.

73-2200. Sram, R. J.; Zudova, Z.; Benes, V.; Symon, K. (Inst. Hyg., Prague, Czechoslovakia). **Induction of translocations in mice by TEPA.** *Newslett. Environ. Mutagen Soc. 3: 13; 1970.* (5 references)

TEPA at 2.5 mg/kg caused 28% translocations in male mice of the F_1 generation developed from germ cells treated in the stage of spermatids. A 2-fold higher frequency of translocations was unexpectedly found in the F_1 males developed from germ cells treated in the spermatogonia stage. The partial sterility observed shows the presence of eucentric interchanges in a stage of spermatogenesis in which the dominant-lethal test reveals no changes. The inadequacy of the dominant-lethal test for evaluating mutagenicity of chemicals is thus demonstrated.

73-2201. Zetterberg, G. (Dept. Genetics Plant Breeding, Royal Agr. College Sweden, 750 07 Uppsala 7, Sweden). **Induction of lysogeny in *Escherichia coli* with analogs of TEPA.** *Newslett. Environ. Mutagen Soc. 3: 14-15; 1970.* (6 references)

TEPA and seven analogs were tested for their ability to induce lysis in lysogenic *E. coli* (λ). The inducing capacity was related to the number of aziridinyl groups. TEPA, which has three aziridinyl groups, was the most effective while those with two were less effective and that with none was ineffective. The inducing capacity is probably due to the alkylating properties of the aziridinyl groups. Strain K39(λ) had more resistance to the toxic effect of the compounds and was induced at a higher frequency than strain K12(λ).

73-2202. Sturelid, S.; Kihlman, B. (Dept. Genetics Plant Breeding, Royal Agr. College Sweden, 750 07 Uppsala 7, Sweden). **Chromosome-breaking effect of TEPA and analogs in bean root tips and in cell cultures of the Chinese hamster.** *Newslett. Environ. Mutagen Soc. 3: 15-16; 1970.* (4 references)

TEPA was a very effective chromosome-breaking agent in both root tips of the broad bean and cell cultures of the Chinese hamster. The same effect was produced in the bean roots and hamster cells with concentrations of $10^{-3}M$ and $3.3 \times 10^{-5}M$, respectively. Chromatid break was the major type of aberration in the hamster cells while these accounted for only 5.9% of the total aberrations in root tips. Most of the TEPA analogs having two aziridinyl groups were as active as TEPA, but the monofunctional analog was 50 times less active in the bean root and 20 times as active in the hamster cells. No activity was observed with the nonalkylating analog HEMPA in either material.

73-2203. De Serres, F. J.; Malling, H. V. (Biol. Div., Oak Ridge Nat. Lab., TN). **Genetic analysis of the *ad-3* mutants of *Neurospora crassa* induced by ethylene dibromide – a commonly used pesticide.** *Newslett. Environ. Mutagen Soc. 3: 36-37; 1970.*

The mutagenicity of ethylene dibromide has been tested in *Neurospora crassa*, using the direct method to screen for purple adenine mutants (*ad-3*). Conidia from a two-component heterokaryon were treated for 3 hr with various concentrations of ethylene dibromide (1.2→1.63 μl) per ml of a 0.06 *M* phosphate buffer adjusted to pH 7.0 containing 10% dimethyl sulfoxide. A total of 1139 *ad-3* mutants were isolated after this treatment with ethylene dibromide; 7 mutants were isolated after treatment with dimethyl sulfoxide alone. Genetic analysis of the mutants was carried out to distinguish point mutations from chromosome deletions and to obtain, at the molecular level, a presumptive identification of the genetic alterations in the point mutations.

73-2204. Malling, H. V.; De Serres, F. J. (Biol. Div., Oak Ridge Nat. Lab., TN). **Captan – a potent fungicide with mutagenic activity.** *Newslett. Environ. Mutagen Soc. 3: 37; 1970.*

The ability of captan to induce forward mutations in the *ad-3* region of a two-component heterokaryon and reverse mutations in a series of tester strains whose mode of reversion represents the most common mutation mechanisms has been tested on *Neurospora crassa*. The forward mutations are induced in the heterokaryotic fraction of conidia of a two-component heterokaryon between biochemically different haploid strains. The *ad-3* mutants are selected by the direct method and can be either point mutations or multilocus deletions (chromosomal deletions). Captan demonstrated mutagenic ability in the forward-mutation system but not in the reverse-mutation system. Genetic characterization of the captan-induced mutants should elucidate the type of mutations induced by this compound.

73-2205. Ficsor, G.; Nii, G. M. (Dept. Biol., Western Michigan Univ., Kalamazoo, MI). **The effect of temperature on the mutagenicity of captan.** *Newslett. Environ. Mutagen Soc. 3: 38; 1970.*

About 1.2×10^8 bacteria, one *lac* mutant of *E. coli* and a *cys* mutant of *S. typhimurium* were plated on

minimal plates. The plates were dried and one 0.05 ml drop of a 1:10 aqueous suspension of a check or previously heat-treated captan (present as a component of the pesticide Tomato Vegetable Dust), was placed on the surface of each plate. Sterile distilled water (0.05 ml) was placed on control plates. The heat treatment of the pesticide was accomplished as follows: a one-to-ten suspension of the pesticide was prepared in sterile distilled water. The suspension was divided into three parts of which one was allowed to stand at room temperature; the second was steam sterilized for 15 min (at about 100°C); and the third was autoclaved for 15 min at 15 pounds pressure (at about 121°C). After cooling the pesticides were placed on the plates. Mutagenic response was determined by counting the number of revertant colonies appearing on the plates in 96 hr at 37°C. The autoclaved pesticide induced 21 times fewer reversions in the *lac* mutant than the pesticide kept at room temperature. The steam-sterilized pesticide induced five times fewer reversions than the room-temperature check. All treatments significantly increased reversions over control levels. The results with the *cys* mutant were similar though less pronounced.

73-2206. Ficsor, G.; Janca, F. C.; Van Hook, IV, C. (Dept. Biol., Western Michigan Univ., Kalamazoo, MI). **Tests for mutagenicity using nitrosoguanidine, DDT, and captan in maize.** *Newslett. Environ. Mutagen Soc. 3: 38; 1970.*

Pollen and seeds from a homozygous stock carrying the three closely linked dominant genes alpha-beta-Sh_2 were treated with captan, DDT, or nitrosoguanidine and crossed with a stock homozygous for the corresponding recessive genes *a-b-*sh_2. Pollen was treated by immersion in mineral oil to which individual test chemicals had been added. Seeds were treated by immersion in either a mineral oil, water, or buffer solution containing individual test chemicals. In mineral oil the seeds failed to germinate. Whole seed and fractional endosperm mutations were stored in the F_1 kernels. F_1 plants derived from nitrosoguanidine pollen treatment were also scored for percent germination and pollen sterility. Of the three compounds tested, only nitrosoguanidine was mutagenic. It exhibited characteristics of X-rays in that it induced only chromosome aberrations, and characteristics of ethyl methylsulfonate by inducing more fractional than whole endosperm mutations. The F_1 seeds derived from nitrosoguanidine-treated pollen exhibited a decrease in germination, indicating the presence of dominant lethals. The high occurrence of pollen sterility in the F_1 plants was an indication of nitrosoguanidine-induced chromosome aberrations. On the basis of F_1 endosperm data pollen treatment appears most effective in inducing mutations.

73-2207. Ficsor, G.; Nii, G. M. (Dept. Biol., Western Michigan Univ., Kalamazoo, MI). **Captan-induced reversions of bacteria.** *Newslett. Environ. Mutagen Soc. 3: 38; 1970.*

Two of fourteen pesticides purchased in a local hardware store were found mutagenic in *E. coli* and *S. typhimurium*. The following technique was used for testing the pesticides: about 1-2 X 10^8 bacteria were plated on minimal plates; the plates were dried and spotted with pesticides and the mutagen nitrosoguanidine. An increase in the number of revertants per plate over control levels or a halo of revertants around the spot of a pesticide or mutagen was taken to indicate a positive mutagenic response. Tomato Vegetable Dust and Ortho Home Orchard Spray were the two mutagenic pesticides; each contains captan as one of their active ingredients. Therefore three components of Tomato Vegetable Dust – methoxychlor, rotenone, and captan – were tested separately, and only captan was mutagenic. In terms of mutagenic effectiveness, captan was most effective when present in Tomato Vegetable Dust, followed by captan with 50% inert ingredients, and captan in Ortho Home Orchard Spray. These differences in effectiveness are probably due to differences in solubility. Among the strains tested, *cys B12* in *S. typhimurium* responded with the highest frequency of reversions, followed by nitrosoguanidine-induced and a nitrogen mustard-induced *lac* mutant of *E. coli*. A 2-aminopurine- and a 5-bromouracil-induced *leu* mutant of *S. typhimurium* did not respond to captan. The *leu* mutants were selected because of their low reversibility and this may account for the lack of positive response. Spontaneous reversion was observed in all five strains; each was revertible by nitrosoguanidine. The *leu* strains had the lowest spontaneous reversion rates and responded the least to nitrosoguanidine.

73-2208. Kilian, D. J.; Benge, M.; Edwards, H. (Dow Chem. Co., Texas Div., Freeport, TX). **Human mutagenic testing of a new organic phosphate insecticide.** *Newslett. Environ. Mutagen Soc. 3: 39; 1970.*

A promising new organic phosphate insecticide, Dowco 179, was tested for human effects. Human exposure via respiratory and skin absorption was increased until such time as the plasma cholinesterase was depressed. A blood lymphocyte culture was prepared for chromosome studies from the volunteer receiving the greatest total exposure.

73-2209. Tessman, I. (Dept. Mol. Biol. Biochem., Univ. California, Irvine, CA 92664). **Induction of transversions and transitions by 1,1-dibromoethane.** *Newslett. Environ. Mutagen Soc. 4: 33; 1971.* (4 references)

In an ochre mutant, induced by hydroxylamine, the number of revertants was increased at least 7-fold over the background level after treatment with 1,1-dibromoethane. Because the wild type and two pseudo-wild types were induced, two different transversions were obtained from the UAA codon, and the DNA bases

affected were limited to T and A. An opal mutant that had been induced from wild type by nitrous acid also reverted to the wild type and two pseudo-wild types after exposure to 1,1-dibromoethane. The mode of action of 1,1-dibromoethane has not been investigated, but A and T must be involved in the transitions and the transversions since neither the ochre codon nor its complement contains G.

73-2210. Zudova, Z.; Sram, R. J. (Inst. Hyg., Prague 10, Czechoslovakia). **Effect of TEPA on the induction of chromosome rearrangements in spermatogonia of mice.** *Newslett. Environ. Mutagen Soc. 4: 41; 1971.*

Results of a study of the effects of a total dose of 4 mg/kg TEPA on spermatocytes in mice corresponded to the results of a study by Leonard and Deknudt with X-rays. The major difference between the results of the two studies was the ratio between the ring and chain forms of chromosome rearrangements. Eightfold more chain forms were observed than ring forms with TEPA, while three fold more translocations in the ring form than in the chain form were found after X-ray treatment. TEPA treatment also resulted in a higher ratio of trivalent-univalent associations.

73-2211. Ficsor, G.; Nii Lo Piccolo, G. M. (Dept. of Biol., Western Michigan Univ., Kalamazoo, MI 49001). **Survey of pesticides for mutagenicity by the bacterial-plate assay method.** *Newslett. Environ. Mutagen Soc. 6: 6-8; 1972.* (12 references)

Of 14 pesticide products tested by the bacterial plate assay for ability to revert two *E. coli lac-* amber mutants to the lactose-fermenting phenotype and a *cys-*and two *leu-*auxotrophs to prototrophy, Tomato Vegetable Dust and Home Orchard Spray gave a positive mutagenic response in the *lac-* and *cys-* strains. Captan, an ingredient common to both products, was subsequently found to be mutagenic when tested with the *cys-* strain. No evidence of mutagenicity was found for a variety of commercial products containing organochlorines, organophosphates, herbicides, and formulation ingredients, Banvel D, atrazine, simazine, Alanap-3 (naptalam), Botran (DCNA), Lorox (Linuron), Cytrol, and Hyvar X (Bromacil) were inactive in the plate assay using *his* auxotrophs, but captan 50W and Tomato Vegetable Dust induced mutations in *his* G 46 and TA 1530 which revert by base substitution like the other strains affected by captan. Several things can explain the failure of pesticides, which induced mutations in barley, to induce reversions in bacteria. DNA backbone breakage, causing light-microscopically detectable chromosome breaks, would not be detected in bacteria. The assay is thus of little value in itself, but when combined with more sophisticated tests can lead to early identification of a mutagenic compound at lower cost.

73-2212. Bridges, B. A.; Mottershead, R. P.; Rothwell, M. A.; Green, M. H. L. (MRC Cell Mutation Unit, Univ. of Sussex, Falmer, Brighton BN1 9QH, Sussex, England). **Captan mutagenesis of repair-deficient strains of *Escherichia coli*.** *Newslett. Environ. Mutagen Soc. 6: 9; 1972.* (4 references)

The mutagenic activity of captan was investigated using various WP 2 trp strains of *E. coli* in simple spot test experiments. A substantial part of the activity was shown to be due to excisable DNA damage mediated by a volatile breakdown product produced at a greater rate under alkaline conditions and probably the result of hydrolysis. Unlike other excisable DNA damage, that produced by the volatile mutagen does not depend on the *Exr*A+ and *Rec*A+ repair functions for its mutagenicity. A nonvolatile diffusable mutagen produced an Exr+-dependent action resulting in excisable damage. The mutagenic properties of captan are essentially those of one or more alkylating agents. The possibility of a carcinogenic hazard from inhaled particles of the fungicide is more serious than that from ingested captan because of the high reactivity of captan in proteinaceous food or blood serum.

73-2213. Kramers, P. G. N.; Knaap, A. G. A. C. (Dept. Radiation Genetics, Univ. Leiden, The Netherlands). **Tests for mutagenic effects of captan in *Drosophila*.** *Newslett. Environ. Mutagen Soc. 6: 26; 1972.*

The induction of recessive sex-linked lethals, II-III translocation, and dominant lethals was investigated in *D. melanogaster* after exposure to captan or orthocide 83, a fungicide containing 83% captan. No significant differences between the treated and control insects were observed in dominant-lethal tests. In a total of 1271 gametes, including post- and premeiotic germ-cell stages, no translocations occurred. Tests for recessive sex-linked lethals revealed only two mutations in 2221 germ cells tested after feeding the insecticide and eight mutations in 2583 postmeiotic germ-cell stages after injection. Delayed effects did not appear in the F_3 generation.

73-2214. Ehrenberg, L. (Radiobiol. Div., Wallenberg Lab., Stockholm Univ., Stockholm, Sweden). **On the mutagenic action of phenoxy acid preparations.** *Newslett. Environ. Mutagen Soc. 6: 27; 1972.*

The National Poisons and Pesticides Board of Sweden appointed a group of experts to evaluate human hazards from the use of 2,4,5-T and 2,4-D in the fall of 1970. The group is reviewing the complex problems involved in estimating possible genetic effect in man from compounds known to be weakly mutagenic in plants. Many formulation components and impurities, as well as decomposition products, are either unknown or untested. The mutagenic activity of tetrachlorodibenzodioxin in *E. coli* and *S. typhimurium* have also been reviewed.

73-2215. Meeter, E. (Med. Biol. Lab. TNO, Rijswijk Z. H., The Netherlands). **Central cholinergic mechanisms in the thermoregulation of the rat.** *Progr. Brain Res. 36: 139-141; 1972.* (3 references)

Sublethal doses of organophosphate ChE inhibitors (like soman) which are capable of passing the blood-brain barrier produce a hypothermia of 4-6° in 2-3 hours in rats during a brief hexobarbitone anaesthesia. Hypothermia can be partially prevented by systemic atropine, and not by atropine methyl nitrate, but spontaneous recovery usually occurs within 12-20 hours. The phenomenon has been observed in mice, and clinical reports of humans poisoned by organophosphates sometimes contain evidence of hypothermia. The guinea pig and rabbit did not respond similarly to administration of organophosphates. Dogs and cats are not expected to exhibit hypothermia. Apparently, the ChE inhibitors shift the set-point for heat release of the hypothalamic thermostat to a lower level which can be restored completely to the normal level. Coupled with other results obtained on hypothermia these findings suggest that cholinergic synapses are involved at a high organizational level in all actions connected with lowering the body temperature in the rat.

73-2216. Avrahami, M.; Gernert, I. L. (Wallaceville Anim. Res. Cent., Dept. Agr., Private Bag, Upper Hutt, New Zealand). **Hexachlorobenzene antagonism to dieldrin storage in adipose tissue of female rats.** *N. Z. J. Agr. Res. 15(4): 783-787; 1972.* (7 references)

Administration of hexachlorobenzene (HCB) at concentrations from 2 to 100 ppm in the diet decreased the deposition of dieldrin in the fat of rats. There was no apparent threshold for the initiation of the effect. Linear correlations were observed between the concentrations of HCB in the fat and the concentrations of HCB in the feed and between the levels of the two organochlorines in the fat. Although young rats accumulated less HCB than adult rats after 10 weeks of dosing, dieldrin storage was half that observed in control animals when HCB residues in the body fat were approximately 330 ppm regardless of the age of the animals. No signs of organochlorine intoxication were observed with any dose of HCB or with the 1 ppm dose of dieldrin, but a slight depression of body weight was observed at higher doses of HCB in growing rats.

73-2217. Metcalf, R. L. (School Life Sci., Univ. Illinois, Urbana, IL). **A model ecosystem for the evaluation of pesticide biodegradability and ecological magnification.** *Outlook Agr. 7(2): 55-59; 1972.* (12 references)

Data obtained from a model ecosystem on ecological magnification and biodegradability of DDT and several asymmetric analogs agrees well with data obtained over the last 20 years in the field. The system consists of an aquarium containing seven liters of water and a sloping shelf of sand. A number of organisms are introduced at different intervals before and after application of the test compound to *Sorghum halepense* on the flattened terrestrial portion. Following application of labeled DDT, DDE made up 52% of the radioactivity in snails, 58% in mosquito larvae, and 54% in fish and was present in *Gambusia* at a concentration 110,000 times that in the water. Studies with methylchlor, methoxychlor, and methiochlor yielded information on the metabolic pathways available in the various test organisms and demonstrated the usefulness of determining accumulation of polar and nonpolar metabolites for predicting biodegradability of pesticides under consideration.

73-2218. Javid, M. Y. (Dept. Biol., Carleton Univ., Ottawa, Canada). **Effect of DDT on temperature selection of some salmonids.** *Pak. J. Sci. Ind. Res. 15(3): 171-176; 1972.* (35 references)

Shifts in temperature selection of three species of salmonids resulted from treatment for 24 hr with sublethal doses of DDT ranging from 2 to 200 ppb. In both the Atlantic salmon and brook trout low doses of DDT generally decreased the selected temperature; higher doses increased it. In rainbow trout exposure to DDT led only to an upward shift in selected temperature. A shock-response to cold temperatures of the gradient was observed in both the Atlantic salmon and brook trout at higher DDT concentrations, while the rainbow trout exhibited a shock-response to warm temperatures. No corresponding shock-response to temperatures higher than 20°C was observed at any DDT concentration. Exposure of Atlantic salmon to potassium cyanide doses ranging from 1 to 200 ppb resulted in a downward shift in selected temperature. The ATP-uncoupler 2,4-dinitrophenol treatment was without any effect on temperature selection of Atlantic salmon. It seemed that DDT-induced changes in the behavior of fish were due to interference by DDT with the thermal acclimation mechanism. It is proposed that increases in metabolic rate lead to response changes associated with warmwards acclimation; decreases in metabolic rate, with coldwards acclimation. DDT is thus visualized as inducing thermal acclimation-like responses by affecting metabolic rate.

73-2219. Jackson, W. B.; Brooks, J. E.; Bowerman, A. M.; Kaukeinen, D. E. (Environ. Studies Cent., Bowling Green State Univ., Bowling Green, OH 43403). **Anticoagulant resistance in Norway rats...in U.S. cities.** *Pest Contr. 41(4): 55, 57, 58, 60-64, 81; 1973.* (15 references)

Results of a survey of Norway rats from U.S. cities participating in the federally funded rat control program indicate that resistance to anticoagulant rodenticides has developed in some urban rat populations. Those cities where numerous resistant rats were found—Chicago, Norfolk, and Washington, D. C.—are areas where anticoagulants have been used for many years. The level of 50% resistant rats, the highest observed, occurred in the

Lawndale section of Chicago were the presence of transients, poor refuse storage and environmental maintenance, and shelter from control efforts in the form of debris or structures contributed to the breeding of resistant animals. At present, anticoagulants are still the rodenticides of choice, but they must be supplemented with poisons like red squill, zinc phosphide, and norbormide at least once a year. Federally funded projects are now stressing environmental sanitation, public education, and community participation in addition to extermination.

73-2220. Balasubramanian, A.; Siddaramappa, R.; Oblisami, G.(Univ. Agr. Sci. Hebbal, Bangalore-24, India). **Effect of simazine and Dithane M-45 on soil microflora and certain soil enzymes.** *Pesticides 7(3): 13; 1973.*

At recommended application rates simazine suppressed the bacterial population, but not the fungal population, in red sandy loam soil. A 10-fold increase in dosage increased this inhibitory effect. Dithane M-45 (maneb), at normal and increased application rates, stimulated the bacterial population and temporarily inhibited the fungal population. It had no effect on the actinomycetes although simazine enhanced this group of microflora. Soil treated with Dithane M-45 evolved more CO_2 than simazine-treated soil, suggesting that simazine was more stable than Dithane M-45.

73-2221. Balasubramanian, A.; Siddaramappa, R.; Oblisami, G. (Univ. Agr. Sci., Bangalore-24, India). **Effect of simazine and Dithane M-45 on nitrification in soil.** *Pesticides 7(3): 14; 1973.*

Soil treated with simazine or Dithane M-45 (mancozeb) at the recommended application rates showed a general decrease in pH over a 60-day incubation period. No significant variation in the ammoniacal-N content was observed. Appreciable accumulation of nitrite-nitrogen occurred after 40 days of incubation with Dithane M-45. More nitrate-N was recovered from simazine-treated soils than from Dithane M-45-treated soils. Simazine at the normal application rate increased the rate of nitrification in soil while Dithane M-45, especially at 10 times the normal rate, inhibited nitrification, resulting in accumulation of considerable nitrite-nitrogen which could inhibit plant growth.

73-2222. Buckland, J. L.; Collins, R. F.; Pullin, E. M. (Res. Lab., May & Baker Ltd., Dagenham, Essex, RM10 7XS, England). **Metabolism of bromoxynil octanoate in growing wheat.** *Pestic. Sci. 4(1): 149-162; 1973.* (1 reference)

[^{14}C]*ring*-Bromoxynil octanoate was applied to the leaves of wheat seedlings which were cultivated in a growth cabinet under controlled conditions for 14 days. Fractionation of the metabolites present in the treated leaves, which accounted for about 63% of the radioactivity applied, indicated a complex metabolic pathway resulting from initial hydrolysis to free bromoxynil, followed by three consecutive or concurrent steps: hydrolysis of the cyano group to the amide and carboxylic acid, followed by decarboxylation to 2,6-dibromophenol (0.5% of the ^{14}C applied); replacement of one or both bromine atoms by hydroxy groups to 3-bromo-4,5-dihydroxybenzonitrile (1.3%) and 3,4,5-trihydroxybenzonitrile (0.6%) or their hydrolysis products; replacement of one or both bromine atoms by hydrogen, giving 3-bromo-4-hydroxybenzonitrile (1.9%) and 4-hydroxybenzonitrile (0.6%) or their hydrolysis products. Some of the phenolic acids or phenols formed are natural plant constituents. The metabolites identified represented in all about 11% of the herbicide applied, but no individual metabolite accounted for more than a small proportion of it.(Author abstract by permission)

73-2223. Stringer, A.; Wright, M. A. (Long Ashton Res. Sta., Univ. of Bristol, Bristol BS18 9AF England). **The effect of benomyl and related compounds on *Lumbricus terrestris* and other earthworms.** *Pestic. Sci. 4(2): 165-170; 1973.* (16 references)

Earthworm populations in apple orchard plots sprayed with benomyl and thiophanate-methyl were greatly reduced. *Lumbricus terrestris*, a surface-feeding species, was not found in the treated plots. Captive worms would not feed on leaf material with spray deposits of benomyl, MBC, and thiophanate-methyl at 1.75 $\mu g/cm^2$, and feeding was significantly reduced when deposits were 0.87 ν-$\beta\omega$2. There was 100% kill of worms in pots of soil within 14 days following a benomyl drench at the rate of 7.75 kg/ha, and a 60% kill following a drench at the rate of 1.55 kg/ha. Worms immersed for 1 min in benomyl suspensions of 0.05 or 0.5% died within 27 and 13 days respectively; those immersed in lower concentrations would not feed for two weeks but eventually did so. It is suggested that the lethal effects of benomyl may be due to anti-cholinesterase activity of the carbamate moiety of the molecule. (Author abstract by permission)

73-2224. Baldwin, M. K.; Davis, R. A.; Burns, D. T. (Shell Res. Ltd., Sittingbourne, Kent, England). **Structural studies and photochemical rearrangement of an animal metabolite of HEOD, the active component of dieldrin.** *Pestic. Sci. 4(2): 227-237; 1973.* (12 references)

To elucidate the mode of formation and possible fate of a major dieldrin metabolite, the structure of 9-hydroxy HEOD was examined by spectroscopic methods and by photolysis. The hydroxyl group was hydrogen bonded to the epoxy group and had a *syn* orientation. The photolysis product was a hydroxylated analog of the previously described photoisomer of HEOD. However, it seems unlikely that the metabolite studied here is a precursor to the other metabolite of

HEOD, the pentachloroketone or Klein's metabolite. (Author abstract by permission, supplemented)

73-2225. Anonymous. **Toxicity hazards of herbicides to livestock.** *Planters Bull. 122: 181-182; 1972.*

Calculations were made from previously established LD50s for various herbicides of the area of vegetation which after spraying would contain the lethal dosage for livestock of three different weights. Sodium arsenite treated vegetation was the most toxic, and dalapon-treated vegetation was the least toxic of those considered. Ingestion of 10 m^2 of vegetation sprayed with paraquat was expected to be lethal to five kilo animals. All calculations were based on the assumption that no degradation had taken place. Using an additive toxicity, the area of vegetation sprayed with combinations of the herbicides which would be lethal were also calculated.

73-2226. Axelsson, U. (Dept. Ophthalmol., Sabbatsberg Hosp., Stockholm, Sweden). **Cataracts following the use of long-acting cholinesterase inhibitors in glaucoma patients. From: The Correlation of Adverse Effects in Man with Observations in Animals.** *Proc. Eur. Soc. Study Drug Toxicity 12: 199-203; 1971.* (23 references)

Retrospective studies on patients treated with long-acting cholinesterase inhibitors for chronic simple glaucoma confirmed the cataractogenic activity of these preparations in man. The incidence of anterior subcapsular vacuoles was distinctly elevated in patients treated with phospholine iodide or Mintacol (paraoxon) for up to three years. Loss of visual acuity also occurred more frequently in patients treated with phospholine iodide or Mintacol than in pilocarpine-treated patients. Decreased vision due to lens changes was particularly apparent in patients over the age of 60 in the phospholine and Mintacol treated groups. Cataracts developed frequently, and also progressed rapidly in eyes with clear lenses when treatment started. Discontinuation of phospholine iodide treatment was followed by regression of lens changes in some patients and progression in others. Local application of 0.25% phospholine iodide or 3% Mintacol solution to the eyes of guinea pigs for up to three years did not cause any type of lens opacity, nor did it alter the spontaneously occurring lens opacities often observed in guinea pigs. Neither x-irradiation nor vitamin C deficiency made the guinea pig lens more vulnerable to cataract changes as a result of phospholine iodide or Mintacol therapy.

73-2227. Rosic, N.; Bignami, G.; Gatti, G. L. (Dept. Therap. Chem., Inst. Superiore Sanita, Rome, Italy). **The use of shuttle-box avoidance in the study of anticholinesterase effects and of anticholinesterase-oxime interactions. From: The Correlation of Adverse Effects in Man with Observations in Animals.** *Proc. Eur. Soc. Study Drug Toxicity 12: 242-246; 1971.* (8 references)

Results of studies on the effects of ChE inhibitors and reactivators on avoidance behavior in rats suggest the need for large scale experimentation on dose levels and timing of treatments, using different conbinations of inhibitors and reactivators. Within a given experiment with physostigmine, low avoidance rats exhibited greater sensitivity to the drug than high avoidance rats. The administration of obidoxime to rats one hour after DFP treatment did not significantly modify the behavioral responses provoked by the ChE inhibitor. Treatment 10 min before and 15 min after DFP administration with 2-PAM or obidoxime did not protect the animals from the behavioral effects of DFP, but did allow a faster partial recovery. Multiple doses of oximes in combination with DFP, parathion, or armine intensified the effects of the ChE inhibitor rather than increasing the antagonistic action of the oxime. The data shows the risks involved in inappropriate dosing and/or improper timing of treatments when reactivators are used to counteract the effects of ChE inhibitors in humans.

73-2228. Zak, F.; Sachsse, K. (Tierfarm AG, Sisseln, Switzerland). **Oral toxicity of the herbicide chlortoluron [*N*-(3-chloro-4-methylphenyl)-*N'*, *N'*-dimethylurea] in rats and dogs. From: The Correlation of Adverse Effects in Man with Observations in Animals.** *Proc. Eur. Soc. Study Drug Toxicity 12: 272-281; 1971.* (20 references)

The acute oral LD50 of chlortoluron was > 5g/kg in dogs of both sexes. In 30- and 90-day feeding studies no adverse effects were observed with a dosage of 600 ppm, but significant weight loss occurred in the 2400 ppm dosage group. All animals died in the 9200 ppm dosage group between the 45th and the 90th day. In the latter group very marked weight depression, mild hemolytic anemia, hemosiderosis of the spleen, brown atrophy, slight hemosiderosis and cholestasis of the liver, and atrophy of the testicles were observed. The acute oral LD50 in rats was > 10 g/kg, and pathological changes observed in 90 day feeding experiments were less obvious than those noted in the dogs.

73-2229. Van Den Bercken, J.; Akkermans, L. M. A. (Inst. Vet. Pharmacol. Toxicol., Univ. Utrecht, The Netherlands). **Temperature and the effect of DDT on the lateral line organ of *Xenopus laevis*.** *IN: Proceedings of First European Biophysics Congress, Volume 5, Verlag Wiener Medizinishcen Akademie, Vienna, 1972, pp. 173-177.* (15 references)

The combined effects of DDT and temperature on a lateral line organ of the clawed toad, *Xenopus laevis*, have been studied. Activity was recorded from one lateral line organ which usually consisted of 5-7 neuromasts and was innervated by two afferent nerve fibers. At room temperature the intervals between two subsequent spikes of the same fiber ranged from 10 msec to more than 100 msec, and the mean rate of spontaneous firing was 30-80 pulses/sec. The rate of discharge

depended directly on temperature, decreasing as temperature decreased. The animals were placed in an aquarium of water to which DDT was added in a concentration of 1-4 ppm. DDT treatment caused spontaneous repetitive activity. Single nerve pulses no longer occurred; all spikes were grouped together in short trains with the number of spikes/train increasing with increased DDT concentration. The effect of DDT on spontaneous activity is highly temperature dependent, with an increase in the number of spikes/train occurring as the temperature decreases. Mechanical stimulation after DDT treatment required a stronger stimulus to induce synchronization of the spike trains with the stimulus and the number of spikes/cycle was constant, not dependent on stimulating freqency as had been the case in mechanical stimulation before DDT treatment. This suggests that DDT does not interfere with the hair cell function, but primarily affects the impulse-generating mechanism in the afferent nerve fiber.

73-2230. Grant, B. F.; Schoettger, R. A. (Fish-Pestic. Res. Lab., Rt. 1, Columbia, MO 65201). **The impact of organochlorine contaminants on physiologic functions in fish.** *Proc. Inst. Environ. Sci. Annu. Tech. Meet. 18: 245-250; 1972.* (14 references)

Recent research activities dealing with chronic biologic effects of organochlorines have indicated that measurement of physiological and biochemical effects of toxicants in fish may be useful in predicting the subtle adverse impact of such materials on aquatic ecosystems. The potential of this tool cannot be fully realized unless effects can be strongly linked to the inability of the fish to overcome normal and abnormal stress, the amounts or rates of contaminant introduced, and the dynamics of the residue within the fish and its habitat. This has been demonstrated in studies of adverse effects on reproduction, blood characteristics, enzyme concentrations, thyroid activity, and other physiologic parameters. Investigations of the uptake of DDT by fish in the laboratory have indicated that when residues of organochlorines in forage fish are similar to or greater than those in the water alone, the forage fish are the major source of these residues in predator fish.

73-2231. Sethunathan, N.; Yoshida, T. (Dept. Soil Microbiol., Central Rice Inst., Cuttack-6, India). **Conversion of parathion to para-nitrophenol by diazinon-degrading bacterium, *Flavobacterium* sp.** *Proc. Inst. Environ. Sci. Annu. Tech. Meet. 18: 255-257; 1972.* (9 references)

Diazinon, parathion, and Dursban (chlorpyrifos) were rapidly degraded to *p*-nitrophenol by the cell-free extract of a *Flavobacterium* sp. isolated from water of a rice field previously treated with diazinon. The extract did not effect the degradation of malathion, which has a P-S-CH bond rather than the P-O-C bond characteristic of the other insecticides. Cells grown in the presence of diazinon or on unamended medium decomposed diazinon and parathion without a lag, indicating that the enzyme involved is constitutive. Para-nitrophenol was not utilized by the bacterium, and no appreciable degradation of aminoparathion was observed although this compound contains the P-O-C.

73-2232. Morton, H. L.; Moffett, J. O. (Plant Sci. Res. Div., Agr. Res. Serv., U.S.D.A., Tucson, AZ 85719). **Effects of herbicides on honey bees.** *Proc. West. Soc. Weed Sci. 25: 15-16; 1972.* (6 references)

Silvex, 2,4-D, 2,4,5-T, 2,4-DB, dicamba, 2,3,6-TBA, chloramben, picloram, EPTC, and dalapon were relatively nontoxic to newly emerged worker honey bees when fed in 60% sucrose at concentrations ranging from 10 to 1000 ppm. Bromoxynil and endothall were toxic at 1000 ppm, and paraquat, MAA, MSMA, DSMA, hexaflurate, and cacodylic acid were extremely toxic at 100 and 1000 ppm. MSMA, cacodylic acid, and paraquat were highly toxic when sprayed on small cages containing adult worker honey bees. Diesel oil or DMSO present in the vehicle carrier increased the toxicity. When applied in a water carrier, 2,4-D, 2,4,5-T, silvex, picloram, endothall, and a mixture of 2,4,5-T and picloram were not toxic. Brood production was not adversely affected when colonies were fed 60% sucrose containing picloram, 2,3,6-TBA, or dicamba at 1000 ppm. At 1000 ppm, however, chloramben and dalapon caused a reduction in brood development, and 2,4-D 2,4,5-T, silvex, 2,4-DB and EPTC markedly reduced or eliminated brood production. At 10 ppm the phenoxy herbicides did not affect brood production, and the removal of the herbicides resulted in normal brood production. Although some toxicity was observed in these experiments, it is more likely that herbicides will injure colonies by depriving them of their food since they usually kill flowers rapidly rather than through direct effects of poisons. 2,4-D and 2,4,5-T were not found in honey sacs of bees or in colonies sprayed by airplanes with these herbicides.

73-2233. Anonymous. **Degree of hazard when using herbicides.** *S. Afr. Sugar J. 57(2): 71; 1973.* (1 reference)

The most useful factor for assessing the potential hazard of a pesticide to humans and domestic animals, although not completely reliable, is the acute toxicity of the chemical. Oral and dermal toxicities according to the LD50 values, are quite high for paraquat, ioxynil, diquat, and some other herbicides while picloram, bromacil, asulam, simazine, dalapon, and diuron have relatively low toxicities. Inhalation toxicity of materials is more difficult to assess than dermal or oral toxicity and is generally less important except in the case of specific fumigants and other volatile materials.

73-2234. Wright, S. J. L. (School Biol. Sci., Bath, Somerset, England). **Degradation of herbicides by soil**

micro-organisms. *Soc. Appl. Bacteriol. Symp. Ser. 1: 233-254; 1971.* (85 references)

For a pesticide to be subject to biodegradation, organisms effective in metabolizing the compound must exist in the soil or be capable of developing there, be in a degradable form, reach the organisms, and induce the formation of enzymes necessary for degradation. Conditions in the environment must favor the proliferation of the organisms and operations of the enzymes. Evidence of microbial degradation has been obtained for most of the herbicides. Phenoxyalkanoates can be degraded by β-oxidation of compounds with long fatty acid moieties, cleavage of the ether link between the side chain and the aromatic ring, and ring hydroxylation prior to attack on the side chain. The formation of nonphytotoxic anilines from phenylcarbamates is apparently a process widely distributed among soil microbes. Detoxication of phenylurea compounds also takes place in soil, and anilines are formed in the process. Hydrolytic dechlorination is the first step in the metabolism of chlorinated aliphatic acids. Substituted benzoates and triazine compounds are quite resistant to attack. Several organisms have been isolated which degrade paraquat and diquat, but these chemicals are strongly adsorbed by some soil particles and are thus unavailable to biological systems. Much of the work on degradation of herbicides has been done with isolated organisms and pure chemicals. This needs to be extended with conditions more closely resembling those found in the field.

73-2235. Cripps, R. E. (Shell Res. Ltd., Borden Microbiol. Lab., Sittingbourne, Kent, England). **The microbial breakdown of pesticides.** *Soc. Appl. Bacteriol. Symp. Ser. 1: 255-266; 1971.* (28 references)

An ether linkage, chlorine atoms, branched carbon chains, or substituted amino groups substituents often found in pesticidal molecules and their residues, increase resistance to microbial degradation by a molecule. The position of substituents also has a marked effect on the rate of degradation. Pure cultures of organisms have been found which will degrade some of the more recalcitrant molecules, suggesting that more research is needed before it will be possible to predict the biodegradability of a compound solely on the basis of its structure.

73-2236. Matsumura, F Boush, G. M. (Dept. Entomol., Univ. Wisconsin, Madison, WI). **Metabolism of insecticides by microorganisms.** *Soil Biochem. 2: 320-336; 1971.* (63 references)

Stable insecticides are most extensively altered by biological degradation systems, particularly microorganisms, in soil. A review of microbial metabolism of insecticides yields too little information on which to base a sound theory explaining such metabolism, but it is possible that their lipophilic properties allow penetration into the normally selective cell wall and that organisms surviving would be either tolerant to or able to degrade the foreign compound to a less toxic or more easily eliminated form. The most extensive studies on DDT metabolites have been involved with the production of DDE and TDE. Probably the best documented metabolic activity of soils has been the epoxidation of cyclodiene insecticides. Organophosphates and carbamates are readily degraded in soil by oxidation and hydrolysis; microorganisms contribute to this degradation. The ease with which insecticide-degrading microorganisms are found is frequently related to the overall stability of the pesticide.

73-2237. Kaufman, D. D.; Blake, J. (Agr. Environ. Qual. Inst., Agr. Res. Cent., Agr. Res. Serv., U.S. Dept. of Agr., Beltsville, MD 20705). **Microbial degradation of several acetamide, acylanilide, carbamate, toluidine, and urea pesticides.** *Soil Biol. Biochem. 5(3): 297-308; 1973.* (21 references)

Microbial degradation of propham, propanil, solan, and swep occurred in enrichment cultures of Celeryville muck or Hagerstown silty clay loam. The degradation in these cultures and in pure cultures of organisms isolated from them was demonstrated by the production of the corresponding aniline, by chloride ion liberation, and by the disappearance of the pesticide. Some of the degradation products were identified. *Fusarium oxysporum* Schlecht, the soil fungus most frequently isolated, possessed the ability to degrade many carbanilates, acylanilides, phenylureas, acetamides, and dithiocarbmates, leading to some detoxication of the pesticides. *Pseudomonas striata* Chester, the most prevalent bacterium, was active in degrading chlorpropham and propham and only slightly active on phenylureas. The oat seedling bioassay indicated not only detoxification of chlorpropham, dicryl, propham, and solan, but also production of some growth promoting substances.

73-2238. Shamiyeh, N. B.; Johnson, L. F. (Dept. Agr. Biol., Univ. Tennessee Agr. Exp. Sta., Knoxville, TN). **Effect of heptachlor on numbers of bacteria, actinomycetes, and fungi in soil.** *Soil Biol. Biochem. 5(3): 309-314; 1973.* (13 references)

Bacteria, of the soil microorganisms tested, were the most sensitive to heptachlor at concentrations of 1, 19, and 25 mg/l. in the culture medium. Some growth retardation occurred with 87% of the 500 bacterial isolates tested, and 63% were inhibited completely on medium containing heptachlor at 25mg/l. Heptachlor, incorporated into the isolation medium at 100 mg/l. prevented the development of 89% of the bacteria, 81% of the actinomycetes, and 50% of the fungi isolated without heptachlor. All of the bacteria that grew on 200 mg/l. heptachlor were Gram-negative; short rods and the fungi were species of *Penicillium, Aspergillus, Fusarium,* and yeast. No actinomycetes developed on this medium. In soybean-alfalfa pots treated with heptachlor and in

treated green house soil an initial depressing effect on fungi and a striking increase in bacterial populations were observed. Higher concentrations of heptachlor in the soil resulted in greater numbers of bacteria. At application rates similar to those used in field practices an increase was observed in heptachlor-resistant fungi, but not resistant bacteria.

73-2239. Foster, R. K.; McKercher, R. B. (Dept. Soil Sci., Univ. Saskatchewan, Saskatoon, Canada). **Laboratory incubation studies of chlorophenoxyacetic acids in chernozemic soils.** *Soil Biol. Biochem. 5(3): 333-337; 1973.* (13 references)

MCPA, 2,4-D, and 2,4,5-T were degraded under laboratory conditions in six Saskatchewan soils. The extent of degradation generally coincided with the size of the microbial population. No degradation was observed in sterilized soil. In most of the soils degradation was most rapid with 50% moisture, and under field moisture conditions the half-life ranged from 14 to 41 days for 2,4-D and MCPA, and was about twice the length for 2,4,5-T. Comparative degradation rates of 1-^{14}C and 2-^{14}-C labeled 2,4-D suggest that degradation was accomplished by ether cleavage rather than decarboxylation.

73-2240. Kacew, S.; Singhal, R. L. (Dept. of Pharmacol., Univ. of Ottawa, Fac. of Med., Ottawa, Canada K1N 6N5). **Metabolic alterations after chronic exposure to α-chlordane.** *Toxicol. Appl. Pharmacol. 24(4): 539-544; 1973.* (18 references)

Daily injection of α-chlordane (5 mg/kg) for 45 days significantly stimulated the activities of pyruvate carboxylase, phosphoenolpyruvate carboxykinase, fructose-1,6-diphosphatase, and glucose-6-phosphatase in liver and kidney cortex of male rats. Statistically significant increases in the quartet of the key, rate-limiting gluconeogenic enzymes in two tissues were noted within 20 days when the daily amount of α-chlordane was raised to 25 mg/kg. Long-term exposure to either dose of the insecticide for 45 days also elevated the concentration of serum urea. The present results together with our previous findings suggest that chronic administration of α-chlordane is as effective as DDT in enhancing the gluconeogenic capacity of both kidney cortex and liver. (Author abstract by permission)

73-2241. Fang, S. C.; Fallin, E.; Montgomery, M. L.; Freed, V. H. (Dept. of Agr. Chem., Oregon State Univ., Corvallis, OR 97331). **The metabolism and distribution of 2,4,5-trichlorophenoxyacetic acid in female rats.** *Toxicol. Appl. Pharmacol. 24(4): 555-563; 1973.* (8 references)

[1-^{14}C]2,4,5-Trichlorophenoxyacetic acid (2,4,5-T) was fed to pregnant and non-pregnant female rats at various dosages, and expired air, urine, feces, internal organs, and tissues were analyzed for radioactivity. During the first 24 hr, 75 ± 7% of the radioactivity was excreted in the urine and 8.2 ± 4.6% in the feces. No ^{14}C was found in the expired air. There was no significant difference in the rate of elimination between the pregnant and nonpregnant rats, or among the dosages used. Radioactivity was detected in all tissues, with the highest concentration being found in the kidney. The maximum concentration of radioactivity in all tissues was generally reached between 6 to 12 hr after po dosing and then started to decline rapidly. Radioactivity was also detected in the fetuses and in the milk. The average biological half-life of 2,4,5-T in the organs was 3.4 hr for the adult rats and 97 hr for the newborn. (Author abstract by permission)

73-2242. Chadwick, R.; Peoples, A.; Cranmer, M. (Environ. Protect. Agency, Perrine Primate Lab., P.O. Box 490, Perrine, FL 33157). **The effect of protein quality and ascorbic acid deficiency on stimulation of hepatic microsomal enzymes in guinea pigs.** *Toxicol. Appl. Pharmacol. 24(4): 603-611; 1973.* (29 references)

Poor quality protein supports and even potentiates some metabolic responses in DDT + lindane-treated guinea pigs. Ascorbic acid deficiency, on the other hand, impairs induction of most of the responses measured. Animals fed soy protein and gelatin exhibit the highest *O*-demethylase activity, hepatic glycogen content and urinary excretion of lindane and lindane metabolites. However, only soy protein permitted significant increases in glucaric acid excretion, hepatic cytochrome P-450 content and glucuronyl transferase. Furthermore, only the guinea pigs on soy protein had statistically significant increases in the metabolic stimulation induced by DDT and lindane in the casein-fed animals. Thus, the animals on the soy protein diet excreted significantly more lindane and lindane metabolites as well as more glucaric acid than the guinea pigs receiving casein. (Author abstract by permission)

73-2243. Sharma, R. P.; Shupe, J. L.; Potter, J. R. (Curriculum in Toxicol., Dept. of Vet. Sci., Utah State Univ., Logan, UT 84322). **Tissue cholinesterase inhibition by 2-carbomethoxy-1-methylvinyl dimethyl phosphate (mevinphos).** *Toxicol. Appl. Pharmacol. 24(4): 645-652; 1973.* (12 references)

Mevinphos inhibited blood, skeletal, and heart muscle cholinesterases following ip injection of sublethal or lethal amounts in rats and rabbits. No inhibition of cholinesterase was noted in brain. The activity in other tissues approached its lowest value in 5-15 min and started recovering afterward. A recovery over the normal amounts of the enzyme was noted in skeletal muscles. Activity nearly approached normal 24 hr after mevinphos administration. Distribution of mevinphos in tissues indicated that the compound entered the brain but the concentrations in brain were considerably lower

than that of blood. In vitro studies indicated that the enzyme preparations from erythrocytes and plasma were inhibited instantly by mevinphos. Brain enzyme required a longer interval to approach maximal inhibition as compared to blood enzymes. Brain enzyme also required higher concentrations of mevinphos for inhibition than did the enzyme from erythrocytes or plasma. Lack of enzyme inhibition in brain in vivo could be due to a combined effect of low concentrations of mevinphos in brain and the differences in the behavior of brain cholinesterase with regard to the inhibitor. (Author abstract by permission)

73-2244. Singleton, S. D.; Murphy, S. D. (Dept. of Physiol., Kresge Cent. for Environ. Health, Harvard Sch. of Pub. Health, Boston, MA 02115). **Propanil (3,4-dichloropropionanilide)-induced methemoglobin formation in mice in relation to acylamidase activity.** *Toxicol. Appl. Pharmacol. 25(1): 20-29; 1973.* (11 references)

Toxic signs after ip doses of 200 to 800 mg/kg of the herbicide propanil in mice included central nervous system depression, loss of reflex, cyanosis and death at the higher doses. Pretreatment with tri-*o*-cresylphosphate (triorthotolyl phosphate, TOTP), an esterase inhibitor, prevented cyanosis but slightly enhanced the CNS depressant actions of propanil. Propanil and a probable hydrolytic metabolite, 3,4-dichloroaniline (DCA), produced methemoglobinemia in mice. Comparison of the time- and dose-response relationships for equimolar doses of propanil and DCA showed that DCA was more potent and had a faster onset of methemoglobin production than propanil. The hydrolysis of propanil to yield 3,4-dichloroaniline by liver homogenates was completely inhibited 18 hr after mice were given 125 mg/kg of TOTP. Methemoglobin formation after propanil was inhibited in mice pretreated with 125 mg/kg of TOTP, but TOTP did not affect DCA-induced methemoglobinemia. Inhibition of propanil-amidase activity and propanil-induced methemoglobinemia had similar TOTP dose- and time-response relationships. Pretreatment with SKF-525A inhibited and phenobarbital pretreatment slightly increased both propanil- and DCA-induced methemoglobin formation. Neither pretreatment affected normal liver propanilamidase activity. These findings indicate that propanil-induced methemoglobinemia is dependent upon the metabolism of propanil by a coupled hydrolytic-oxidase enzyme system. The acute toxicity-TOTP interaction experiments indicate that methemoglobinemia can account for the cyanotic appearance of propanil-poisoned mice, but that methemoglobin formation is apparently not responsible for loss of righting reflex and death. (Author abstract by permission)

73-2245. Webb, R. E.; Hartgrove, R. W.; Randolph, W. C.; Petrella, V. J.; Hortsfall, Jr., F. (Dept. of Biochem. and Nutr. and Dept. of Hort., Virginia Polytechnic Inst. and State Univ., Blacksburg, VA 24061). **Toxicity studies in endrin-susceptible and resistant strains of pine mice.** *Toxicol. Appl. Pharmacol. 25(1): 42-47; 1973.* (12 references)

Oral LD50 studies were conducted in strains of endrin-susceptible and resistant pine mice live-trapped in different apple orchards. The degree of resistance to endrin decreased in the orchard once endrin application as a control measure was terminated. Endrin-resistant mice showed a 2-fold degree of cross-resistance to dieldrin and an absence of cross-resistance to the organophosphate Gophacide (*O,O*-bis(*p*-chlorophenyl)-acetimidoylphosphoramidothioate) and to the indirect anticoagulant chlorophacinone. Both Gophacide and chlorophacinone were more lethal to the endrin-resistant mice. Endrin toxicity studies in offspring indicated the resistance is heritable in first generation progeny. (Author abstract by permission)

73-2246. Chhabra, R. S.; Fouts, J. R. (Pharmacol. and Toxicol. Branch, Nat. Inst. of Environ. Sci., NIH P.O. Box 12233, Research Triangle Park, NC 27709). **Stimulation of hepatic microsomal drug-metabolizing enzymes in mice by 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane (DDT) and 3,4-benzpyrene.** *Toxicol. Appl. Pharmacol. 25(1): 60-70; 1973.* (24 references)

In previous reports from this laboratory it was shown that DDT or benzpyrene apparently did not stimulate hepatic drug metabolism in mice. Other investigators have recently reported that mice can respond to DDT or benzpyrene treatment by exhibiting increased levels of hepatic drug metabolizing enzymes. We reinvestigated the effect of DDT or benzpyrene on the strain of mice we had previously used. We found that apparent resistance of mice to hepatic microsomal enzyme induction by DDT or benzpyrene was related to the doses of DDT or benzpyrene used, the duration of treatment with these inducers, and the short-lived duration of the induction. (Author abstract by permission)

73-2247. Kibler, W. B. (Dept of Neurol., Vanderbilt Univ. Sch. of Med., Nashville, TN 37232). **Skeletal muscle necrosis secondary to parathion.** *Toxicol. Appl. Pharmacol. 25(1): 117-122; 1973.*

Twenty-six Sprague-Dawley rats were given daily ip injections of parathion for two weeks and sacrificed at regular intervals. The skeletal muscle necrosis is believed to be due to excessive acetylcholine. Adaptation is rapidly developed; this may be the result of progressive refractoriness of the end plate. (Author abstract by permission)

73-2248. Wainwright, S. D.; Wainwright, L. K.; Fraser, I. H. (Biol. Dept., Mount St. Vincent Univ., Halifax. Nova Scotia, Canada). **Hemoglobin formation in the explanted chick blastodisc; a model system for evalua-**

tion of embryotoxic agents as illustrated by effects of vinblastine, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)-ethane (DDT) and sodium diethylbarbiturate. *Toxicol. Appl. Pharmacol. 25(1): 123-129; 1973.* (12 references)

Concentrations of $4 \times 10^{-6} M$ vinblastine and 5 μg/ml ($1.77 \times 10^{-4} M$) DDT markedly inhibited formation of hemoglobin in the blood islands of explanted blastodiscs exposed to the agent before attaining the 6-somite stage of development. More advanced blastodiscs were less sensitive to inhibition by either vinblastine or DDT. Low concentrations of sodium diethylbarbiturate ($10^{-6} M$) markedly stimulated hemoglobin formation at all developmental stages examined. (Author abstract by permission)

73-2249. Haufe, W. O. (Vet. Med. Entomol. Sect., Research Station, Canada Dept. Agr., Lethbridge, Alberta, Canada T1J 4B1). **Interaction of pesticidal toxicity, parasites, and reversible anticholinesterase activity as stresses on growth rate in cattle infested with horn flies *Haematobia irritans* L.** *Toxicol. Appl. Pharmacol. 25(1): 130-144; 1973.*

Differential rates of gain in groups of immature cattle exposed to horn fly attack were used to distinguish the interaction of pesticidal activity and nonpesticidal pharmacologically induced stress with host-parasite reaction. Group treatments included DDT and coumaphos sprays as pesticidal agents, which produce low and moderate bovine toxicities, respectively, at parasiticidal concentrations; water spray at high pressure as an intermittent but relatively innocuous physical suppressor of infestations; and eserine as an agent of intermittent pharmacologically induced stress superimposed on uncontrolled fly attack. Feeding activity of parasites retarded growth of hosts during the buildup but not during the decline of the infestation. Toxicity of coumaphos applied as a prophylactic treatment reduced potential for accelerated growth, especially in the compensatory period during and after decline in infestations. Anticholinergic stress induced by eserine amplified parasitic retardation of growth in unprotected animals for 6-9 weeks, after which animals adapted to the drug and corresponded more closely in weight with the untreated group. Intermittent reduction of flies with a water spray induced a high, stable rate of growth during the horn fly season. With DDT-protected animals as a baseline, growth rate under pressure of infestation was retarded by interacting stresses at the rate of 1, 8, 15.3, and 17.5 equivalent growing days, respectively, for animals subjected to water treatment, coumaphos spray, no treatment, and pharmacologically induced stress during a 98-day observation period.

See also

73-2034	73-2062	73-2078	73-2090	73-2106	73-2135	73-2147
73-2035	73-2066	73-2081	73-2099	73-2116	73-2143	73-2263
73-2038	73-2074					

73-2250. Ballinger, D. G. (Anal. Qual.Contr. Lab., Environ. Protection Agency, Cincinnati, OH). **Laboratory methods for the measurement of pollutants in water and waste effluents.** *American Institute of Aeronautics and Astronautics, paper no. 71-1033, New York, 1971, 18 p.* (18 references)

Available methods for determining pesticides in water and wastewater are generally specific and sensitive, but precision is poor and the methods are slow and not easily automated. Metal analysis is usually precise, specific, sensitive, and easily automated while the speed of various procedures varies. Although most metals can be determined by atomic absorption, emission spectroscopy, or polarography, arsenic still requires colorimetric procedures. Often several techniques are required for identification of pesticides because of the similarity of compounds within a group. Gas chromatographic techniques are the most frequently used and often combined with mass spectrometry, IR spectrometry, solubility separations, and TLC. Precision and accuracy are particularly important in water and waste laboratories because of the need for data which can be compared in litigation. (Presented at Joint Conference on Sensing Environmental Pollutants, Nov. 8-10, 1971, Palo Alto, CA)

73-2251. Mancy, K. H.; Weber, Jr., W. J. (Univ. Michigan, Ann Arbor, MI). **Industrial toxicology and its control.** *IN: Analysis of Industrial Wastewaters, John Wiley & Sons, Inc., New York, 1972, pp. 493-496.*

Considerable research has been done to devise methods for the separation and detection of traces of pesticides, which are toxic in small quantities and often persist and accumulate in the environment. Concentration and separation of these compounds from large volumes of natural waters and wastewaters are generally achieved by liquid-liquid extraction or adsorption on carbon. Spectrophotometric analysis, elemental analysis, measurement of biological activity, and chromatographic analysis are used for identification of the residues. Particularly high sensitivity can be obtained with electron capture GC while microcoulometric titrator detectors provide high specificity for halogens, sulfur, and phosphorus. Comparison of IR spectra of material separated chromatographically provides an excellent system of identification.

73-2252. Frei, R. W.; Lawrence, J. F.; LeGay, D. S. (Trace Analysis Res. Cent., Dept. of Chem., Dalhousie Univ., Halifax, Nova Scotia, Canada). **The analysis of carbamate and urea herbicides by fluorimetry of their dansylated amine moieties.** *Analyst 98(1162): 9-18; 1973.* (24 references)

Carbamate and urea herbicides have been analysed by thin-layer chromatography and fluorimetry of the 1-dimethylaminophthalene-5-sulphonyl (dansyl) derivatives of their aniline hydrolysis products. The herbicides were hydrolysed with 1 *M* sodium hydroxide solution at 80°C for 40 minutes and then the liberated anilines and amines were extracted into hexane. Aliquots of the hexane solution were spotted onto a chromatographic plate and made to react in situ with at least a ten-fold excess of dansyl chloride, which was spotted over them. Next, the plate was sprayed until moist with a 20 per cent solution of triethanolamine in propan-2-ol and the spots were analysed fluorimetrically in situ. The hydrolysis and coupling reaction of the herbicides, fluorescence phenomena and chromatographic properties of the derivatives were investigated. Detection limits of 1 ng per spot taken at a 3:1 signal to noise ratio and a reproducibility of 3 to 5 per cent relative standard deviation can be expected and the calibration graphs are linear up to 300 ng per spot. Natural water samples can be analysed to determine concentrations of herbicides of a few parts per billion (10^9) with high recoveries and without the necessity of a clean-up step. (Author abstract by permission)

73-2253. Panel on Malathion and Dichlorvos Residues in Grain (Comm. Anal. Meth. for Residues of Pestic. and Vet. Products in Foodstuffs, Min. of Agr., Fish. and Food, Plant Pathol. lab., Harpenden, Herts., England). **The determination of malathion and dichlorvos residues on grain.** *Analyst 98(1162): 19-24; 1973.* (4 references)

A study of solvents for extraction of malathion and dichlorvos from grain revealed that methanol extraction without cleanup is optimal. In this study, after addition of the pesticide solution to grain, the grain was stored for 24 hr in a deepfreeze to permit pesticide penetration. The recommended analytical determination comprises grinding the undried grain to a powder, taking care not to overheat the sample, and homogenizing 20 g of the powder with 40 ml of methanol for 2 min. The homogenate is filtered through a Buchner funnel, returned to the homogenizer, and rewashed twice with methanol. The combined filtrates are concentrated to 2 ml at a temperature below 35°C, then diluted to the desired volume with acetone. A phosphorus-sensitive thermionic detector or a flame-photometric detector may be used for gas chromatography of the solutions. A number of suitable chromatographic columns, many of the succinate ester type, and conditions are listed. Recoveries of malathion from spiked wheat using this method averaged nearly 90%.

73-2254. Greve, P. A. (Rijks Inst. voor de Volksgezondheit, Utrecht, The Netherlands). **Toxische organische microverontreinigingen in oppervlaktewater. [Toxic organic trace pollutants in surface water.]** *Chem. Weekblad 41(68): 11, 13, 15; 1972.* (5 references) (Dutch)

During the analysis of trace pollutants in water, group tests are supplemented by tests for specific substances. Pollution analyses are aimed at detecting substances or groups of substances which are known to be

harmful and which can be expected to be present in the water. The total organic material content of the water can be broken down into hetero-element groups, such as organics containing halogen, sulfur, nitrogen, or phosphorus. Microcoulometric methods are available for organically bound chlorine and sulfur in surface water, and a conductometric determination for nitrogen is under development. At the next level of specificity group reactions can be used to detect chemical groups; colorimetric tests are used for the dichiocarbamates; groups with specific actions such as enzyme inhibition tests are used for the cholinesterase inhibitors. A group reaction is not commonly used for the organochlorine insecticides; they can be determined individually and simultaneously by gas chromatography with a mass spectrometric detector. Carbaryl, dimethoate, diazinon, malathion, and parathion were identified in Rhine water at 0.01-0.1 ppb. Rhine water in 1969-1972 contained average values of 0.15 ppb α-BHC, 0.01 ppb γ-BHC (lindane), and 0.13 ppb HCB.

73-2255. Baluja, G.; Franco, J. M. (Inst. Gen. Organic Chem., Madrid 6, Spain). **Thermal decomposition of labile chlorinated pesticides and efficiency of mixed stationary phases in gas-liquid chromatography analysis.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 263-272.* (9 references)

Efficiency and performance in the GLC analysis of common chlorinated pesticide residues, using mixed liquid phases in low-loading columns of medium polarity, have been investigated. The liquid phases used were 2% Oronite Polybutene 128/1.5% OV-17, 1.5% OV-17/1.95% QF-1, 2% Oronite Polybutene 128/1.95% QF-1, 2% Oronite Polybutene 128 (.02% Epikote 1001), 5% DC- 200/7.5% QF-1, and 9.9% DC-200. Retention times did not vary much from published lists. Endrin gave at least three main peaks with very different retention times, suggesting isomerization. This isomerization was very marked on polar columns, decreasing in non-polar phases. Isomerization also increased with increasing temperatures. Thermal decomposition of DDT followed the same pattern to a lesser degree. It is necessary to determine whether TDE and DDT derived compounds detected in the sample by the chromatogram are present due to thermal processes in the column or arose from a metabolic evolution before extraction of the organ or tissue under study. No decomposition of other common chlorinated pesticides was observed. Higher temperatures may improve the efficiency with which pairs of compounds of similar retention times are separated. Although endrin and DDT undergo less thermal degradation on the non-polar column, use of either 2% Oronite Polybutene 128 1.95% QF-1 or 5% DC-200 12,500 cst/7.5% QF-1 affords a broader spectrum of relative retention times, thus increasing the efficiency of separation.

73-2256. Tietz, H. (Farbenfabriken Bayer Ag, Leverkusen, Germany). **Chemistry and metabolism of terminal residues of fungicides.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 347-366.* (60 references)

A review of fungicide development is presented, starting in the 19th century. A listing of various analytical tools useful for the study of metabolism and terminal residues of three classes of fungicides begins with the dithiocarbamates. These fungicides are best studied by breaking down the compounds to amine and carbon disulfide and determining the latter colorimetrically as a copper dithiocarbamate complex. This method will detect surface residues. Paper chromatographic procedures are available to identify dithiocarbamates and degradation products. The terminal residues of phthalimides and related compounds, mainly captan, folpet, Difolatan (captafol), and dichlofluanid are detected by reaction with resorcinol, reaction with pyridine, polarography, or an infrared method. Benomyl may be determined by either a fluorometric or a colorimetric method, for both of which it must be converted to 2-aminobenzimidazole. Even with the variety of analytical tools available, the significance of an analytical predication depends on sufficiently detailed metabolic studies.

73-2257. Winteringham, F. P. W. (Joint FAO/IAEA Div., Vienna, Germany). **The current status of radioactive tracer techniques for studying pesticide residue problems.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp 367-378.* (28 references)

Radioactive tracer techniques are useful in monitoring the nature, extent, and significance of pesticide residues. Radioassay affords immediacy and accuracy often without need for sample destruction. The continuing residue loss through harvest, storage, washing, milling, and other processing steps can be followed quantitatively without subjecting samples to solvent extraction and clean-up. Radioassay of extracts and extracted fractions yields information concerning completeness of recovery and possible chemical binding of residues. Residues can be rapidly characterized as parent pesticides or various specific metabolites. A labeled pesticide can be followed in the presence of background levels of other pesticides. Labeled metabolites may be identified below the limits of chemical detection. There are four main considerations in conducting a sucessful tracer experiment: availability of suitably labeled pesticides; effective extraction fractionation and radioasssay procedures; careful identification of recovered labeled derivatives; and correct experimental design and data interpretation.

73-2258. Koivistoinen, P. E.; Koskine, E. H.; Kansala, A.; Salminen, K. (Inst. Food Chem. Technol., Univ. Helsinki, Helsinki, Finland). **A double-label isotope technique for studying the degradation of pesticides: applica-**

tion to malathion. *IN: Fate of Pesticides in Evironment, Gordon and Breach, London, 1972, pp. 379-388.*

The phosphorus atom of malathion was labeled with ^{32}P and served as a molecule-bound internal standard, allowing conclusions to be drawn on the chemical structure of the derivatives of the partner molecule. Different groups of the molecule were labeled with ^{35}S, ^{14}C, and ^{3}H. The degradation products after acid hydrolysis were removed by ion exchange chromatography. Absolute activities of different isotopes were assayed by liquid scintillation counting. Changes in ratio of radioactivities of tracer used to ^{32}P provide information concerning chemical structures of degradation products and overlapping of compounds in the effluent fractions. Pathways of chemical degradation can be postulated and the validity of ion exchange chromatography for separating degradation products evaluated. It is emphasized that the group ratio will indicate only the presence of the labeled groups or the quantity removed. If the molecule has a number of similarly labeled groups in various positions, the group ratio will not indicate which groups are absent; neither will it demonstrate what chemical changes have occurred in other parts of the molecule.

73-2259. Brooks, G. T. (Agr. Res. Council, Unit Invertebrate Chem. Physiol., Univ. Sussex, Brighton, England). **The preparation of isotopically labelled pesticides—principles and problems.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 405-418.* (15 references)

Use of a labeled intermediate common to a series and replacement of a readily detached peripheral group of a complex molecule by a labeled moiety are two frequently used and easily accomplished methods for synthesizing isotope labeled pesticides. The successful use of labeled moieties placed into the chemical structure of the pesticide depends on the nature of the investigation since these added moieties are frequently lost in biological systems through enzyme attack. Some of the problems encountered when no alternative to labeling the central skeleton of a complex molecule exists include: labeling of two different elements; labeling in different ring systems; the requirement of different synthetic routes for different isomers; and all problems associated with multi-stage organic synthesis on a small scale.

73-2260. Disney, R. W. (Ministry of Agr., Pest Infestation Control Lab., Buckinghamshire, England). **Spark chambers for visualization of activity on radiochromatograms.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 419-421.* (4 references)

Scanning techniques for determining radionuclide distribution on chromatograms are time consuming and cannot simultaneously achieve both high resolution and counting accuracy. A spark chamber has been described in which the chromatogram is placed in such a way that the presence of radioactivity causes photographable localized sparking along the ionized tracks left by charged particles escaping from the chromatogram. Two types of chambers exist-one with crossed-wire construction and the other with parallel spiral cathodes with coaxial anodes. Each type operates with a fixed applied potential. Thus, each spark completely discharges the chamber which must then recharge. Consequently, the chamber can respond to only one event at a time and has a long dead-time.

73-2261. Disney, R. W.; Fowler, K. S. (Ministry of Agr., Pest Infestation Control Lab., Buckinghamshire, England). **Phosphorus-32-labelled phosphine in the determination of fumigation residues in grain.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 423-425,* (7 references)

A direct method is described for the determination of residual phosphorus compounds in grain after prolonged aeration. The method is based on the use of ^{32}P. Fumigated wheat (3.0 mg/l. in air, 25°C) was thoroughly aired in a stream of nitrogen until no further significant amount of radioactivity could be removed. Aliquots were then digested to give a clear colorless solution for Cerenkov counting in a liquid-scintillation counter. The residue value calculated as phosphorus after five days' exposure was 1.2 ppm when the moisture content of the wheat was 10.0% wet weight, increasing to 7.3 ppm as the moisture content increased to 19.7%. After 14 days' exposure the residue was 2.9 ppm when the moisture content was 10.0%, increasing to 18.4 ppm at moisture content of 19.7%. The location of the residues was studied in the 12.4% moisture content grain and was shown mostly to be in the outer layers and in the crease.

73-2262. Lubkowitz, J.; Parker, W. C. (Dept. Nucl. Chem., Venezuelan Inst. Sci. Invest. Caracas, Venezuela). **Recent advances in the development of ionization detectors for non-radioactive and radioactive fractions from GLC columns.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 427-429.*

An electron capture detector which uses 8 mCi ^{147}Pm as the source instead of ^{63}Ni has been developed for use in the saturation current region for the detection of non-labeled compounds. It can also be heated to high temperatures, and the cost of the ^{147}Pm source is considerably lower. The shorter half-life of ^{147}Pm is not viewed as a disadvantage. Tests have been conducted with 0.1-50 ng of aldrin, dieldrin, DDT, and lindane. Geiger counter detectors have been designed which operate with a constant external source of ^{60}Co of approximately 10 μCi. Since the detector operates in the Geiger region of gas amplification, only low activities are required. The counter is a concentration sensitive device operating in three regions corresponding to conditions of electron impact, photoionization, and complete dis-

charge. Results of sensitivity tests were as follows: lindane 1.7×10^{-12} moles/sec, DDT 3.2×10^{-12} moles/sec, and aldrin 2.6×10^{-12} moles/sec. It has been used at temperatures as high as 200°C, with no effect on the response. Compounds labeled with ^{14}C can also be analyzed.

73-2263. Korte, F. (Org. Chem. Inst., Bonn Univ., Bonn, Germany). **Analytical problems in the measurement of pesticide residues.** *IN: Fate of Pesticides in Environment, Gordon and Breach, London, 1972, pp. 465-477.* (21 references)

In measuring pesticide residues analytical data older than a few years or not verified by independent methods should not be trusted. Not only must the parent compounds be subject to analysis, but the conversion products as well. Collection, storage, extraction, and clean-up of representative samples are difficult. Only after an intensified extraction procedure yields no further increase in the amount of pesticide extracted can the procedure be considered adequate. Solvents which coagulate proteins should only be used in the last extraction step. New methods are needed for the recovery of hydrolytic degradation products. Quantitative determination of pesticide residues may be carried out with fairly good results by GLC. Important progress in TLC analysis may be forthcoming from a new scanning microdensitometer equipped with a xenon or xenon-mercury lamp and a monochromator which allows measurements in the range of 200 to 700 mμ. This will enable many substances to be determined on TLC without visualization with spray reagents. The claim that cholinesterase inhibition is a measure of the toxic effect of organophosphate and carbamate insecticides is under attack as an oversimplification. The majority of current analytical methods developed measure on the inhibition of the pseudocholinesterases, which are located in the plasma of most animals. These methods may be useful, especially for screening purposes, to reveal the presence of inhibitors, but must not be regarded as indices of total residue toxicity. Unless identity of response between red blood cell cholinesterase and the esterases of brain and nervous tissue is established, even the determination of red blood cell cholinesterase inhibition will remain unacceptable for toxicologic evaluation of a residue.

73-2264. Seawright, J. A.; Bowman, M. C.; Lofgren, C. S. (Insects Affecting Man and Animals Res. Lab., Agr. Res. Serv. U.S. Dept. of Agr., Gainesville, FL 32601). **Insect chemosterilants: gas chromatography, p-values, and relationship of p-values to sterilant activity in pupae of *Anopheles albimanus*.** *J. Econ. Entomol. 66(3): 613-617; 1973.* (15 references)

Relative retention times of 19 insect chemosterilants on 6 columns were determined using a gas-liquid chromatograph with temperature programing and a flame photometric detector. The compounds containing a P-S moiety and apholate chromatographed well on all columns, whereas those with a P-O group tended to "tail" (give nonsymmetrical peaks) on all columns except the most polar one (OV-225). The OV-225 column is therefore recommended for analyzing all compounds except apholate. The Dexsil 300 phase is preferred in the latter case because of its high thermal stability since apholate is analyzed at a much higher temperature than the other compounds. To obtain maximum sensitivity and reproducibility in the determination of trace quantities of chemosterilants it is necessary to condition the column to the substrate and compound being analyzed. The p-values (partitioning fractions) were also determined for these 19 compounds in 11 solvent systems. These values indicate the relative polarities of the compounds and are useful in developing extraction methods as well as for confirming identities of unknowns. Low polarity was associated with high chemosterilant activity.

73-2265. Struble, D. L.; McDonald, S. (Res. Sta., Canada Dept. of Agr., Lethbridge, Alberta, Canada T1J 4B1). **Residue analysis of chlorpyrifos and its oxygen analogue in field-treated wheat plants.** *J. Econ. Entomol. 66(3): 769-772; 1973.* (14 references)

Methods were developed for analyzing chlorpyrifos and its oxygen analog in wheat plants and kernels. The recoveries from fortified plant samples ranged from 85 to 95% for chlorpyrifos and from 58 to 98% for the oxygen analog (*O,O*-diethyl *O*-3,5,6-trichloro-2-pyridyl phosphate). Wheat plants at the early-tillering stage were field treated with chlorpyrifos emulsifiable concentrate at 0.56 and 1.12 kg AI/ha. Residues decreased steadily throughout the growth period. Straw harvested 63 days after treatment contained 0.003 ppm or less chlorpyrifos, and the oxygen analog was not detectable (<0.005 ppm). Residues were not detectable in the wheat kernels. The dilution of the residues by plant growth was calculated, and the actual disappearance of chlorpyrifos was estimated. (Author abstract by permission)

73-2266. Jarosch, R. (Univ. Wisconsin Center-Sheboygan, Sheboygan WI 53081). **The determination of pesticide residues. A laboratory experiment.** *J. Chem. Educ. 50(7): 507-508; 1973.* (5 references)

A basic procedure for the determination of pesticides in water, soil, and aquatic animals has been worked out for use as a student experiment in Organic Chemistry. Extraction was carried out using 15% ethyl ether-hexane for most samples and hexane or hexane-acetone for fish. Separation from waxes, oils, and other interfering substances was achieved using a Florisil column. The pesticides were determined qualitatively on silica gel TLC plates using carbon tetrachloride or other low polarity solvents for carriers and Rhodamine B for visualization reagent. Quantitative determination

requires a gas chromatograph equipped with an electron capture detector, apparatus generally too expensive for an undergraduate laboratory. Dieldrin, endrin, lindane, TDE, heptachlor, DDT, DDE, and aldrin were separated by this method.

73-2267. Ishii, T. (Hyogo Prefect. Environ. Protect. Res. Inst., Japan). **[Polarographic analysis of air pollutants. 2. Analysis of organic materials.]** *Kogai To Taisaku (J. Pub. Nuisance) 8(7): 665-672; 1972.* (65 references) (Japanese)

Polarography as an analytical method for various air pollutants is reviewed with reference to previously published material. Among the eight isomers of BHC, only gamma-BHC (lindane) responds to polarography with reduction waves. A standard procedure for measuring BHC is presented. Polarography has been successfully applied for measuring trace amounts of lindane aerosol in the air. Lindane is collected by passing polluted air through a glass impinger filled with 75% ethanol. Sorensen buffer and sodium chloride are added to a quantity of lindane-alcohol solution. The alcohol concentration of the polorographic solution is 50%. Polarography is also applied to DDT analysis. The oscillopolarographic quantitation limits of various organophosphate insecticides and regulating solutions, samples of oscillopolarograms, BHC polarogram samples, and half-wave potentials of DDT and related compounds are illustrated.

73-2268. Yuki, F.; Nagase, M.; Murano, A. (Takarazuka Res. Lab., Sumitomo Chem. Ind. Co. Ltd., Takarazuka, Japan). **[Simultaneous determination of organophosphorus pesticides in mixed commercial formation by gas chromatography.]** *Noyaku Seisan Gijutsu (Pestic. Tech.) 30: 15-21; 1973.* (9 references) (Japanese)

Retention times (RTs) and conditions for the gas chromatography of 17 kinds of organophosphorus pesticides (OPs) with various types of chemical structures on 12 column packings have been determined using a hydrogen flame ionization detector. Relative retention times (RRTs) of the 17 OPs on each column packing were calculated using fenitrothion as a standard. On an Apiezon L column which showed a fairly large range of RRT for the 17 OPs and also fairly favorable separation, trials of simultaneous determination of each component in seven commercial formations (EC, dust, and granule) containing two OPs each were carried out using fenitrothion or Papthion (phenthoate) as an internal standard. The analytical values obtained from the trials on the commercial formulations agreed with the calculated ones, and these OPs in the mixed formulations were accurately determined. It was difficult to separate all of the OPs on only one column; however, it would be possible to determine OPs by a favorable choice of columns for separation and by the use of an adequate OP as an internal standard.

73-2269. Takase, I.; Osuga, S. (Agr. Chem. Inst. Nihon Tokushu Noyaku Seizo Co. Ltd., Tokyo, Japan). **[Gas chromatographic micro-determination of pesticides. Part 6. Determination of some residual *N*-methylcarbamate insecticides as 2,4-dinitrophenyl ether derivatives.]** *Noyaku Seisan Gijutsu (Pestic. Tech.) 30: 22-28; 1973.* (7 references) (Japanese)

A method for the analysis of residues of insecticidal *N*-methylcarbamates was studied. The method involves extracting the residue from specimens, hydrolyzing the residue to a phenol and reacting the phenol with 2,4-dinitrofluorobenzene to form the corresponding 2,4-dinitrophenyl ether derivative. The ether is cleaned up and detected by gas chromatography with an electron capture detector. The application of this method for determining *N*-methylcarbamate residues in rice grain and soil was investigated. Recoveries of *o*-isopropoxyphenyl, *m*-tolyl, 3,4-xylyl, and *o*-sec-butylphenyl *N*-methylcarbamate added at levels of 0.1 and 1 ppm to rice grain were 80%. Recovery of pesticide added to soil at a level of 0.2 ppm was more than 90%. The limit of detection was 0.002 ppm. A glass column packed with 10% Silicone DC-200 on 147-175 micron Gaschrom Q was utilized in the present gas-chromatographic study. The method described could be used for simultaneous determination of several *N*-methylcarbamate insecticides by selection or combination of suitable GLC columns and operating conditions.

73-2270. Agricultural Chemicals Inspection Station (Tokyo, Japan). **[Official methods of analyzing agricultural chemicals. Part 24. Granular formulations of ethylthiometon (disulfoton).]** *Noyaku Seisan Gijutsu (Pestic. Tech.) 30: 37-38; 1973.* (1 reference) (Japanese)

The official method for analyzing ethylthiometon granular formulation, containing nominally 5% of disulfoton as the a.i., was published May 20, 1972, by the Japan Ministry of Agriculture and Forestry as method No. 776. A specimen containing about 50.0 mg a.i. is extracted for one hour with 100 ml of chloroform with shaking; the chloroform is evaporated to less than 10 ml, and the sample made up to 50 ml again by addition of chloroform. A 1 ml aliquot of the solution is transferred to a silica plate and developed upwards for 12 cm with a mixture of hexane and acetone (10:1 v/v). The part of the developed plate which shows a dark purple color under UV irradiation is scraped off and dissolved in methanol. The methanolic solution is made up to 50 ml. A 20 ml aliquot of the solution is heated slowly to evaporate the solvent, and after accurate addition of 2 ml of 50% sulfuric acid then 5 ml of nitric acid the mixture is heated on an electric heater. When the evolution of brown fumes has ended, the heating is continued until white fumes of sulfuric acid evolve. Five ml of water is added to the nitrated solution, and it is heated for 2 min. The solution is made up to 30 ml by adding water; 5 ml of vanadate reagent and 5 ml of molybdate reagent are added in that order. The solution is made up

to 50 ml with water and left standing at room temperature for 15 min. The extinction of the solution at 420 nm is read, and the blank value subtracted. Ethylthiometon content is calculated from a calibration curve. Commercial products labeled as 5% ethylthiometon granules contained 5.35-5.45% disulfoton averaging 5.41%.

73-2271. Analytical Quality Control Laboratory (Environ. Protect. Agency, Nat. Environ. Res. Cent., Cincinnati, OH 45268). **Methods for organic pesticides in water and wastewater. 1971.** *U. S. Govt. Printing Office, Washington, 1972, 58 pp. (41 references)*

A method prepared by the Analytical Quality Control Laboratory for the determination of organochlorine pesticides is recommended for all laboratories monitoring these materials in waters and wastewaters sampled by the Environmental Protection Agency. The method includes several analytical alternatives, the use of which depends on the nature and extent of interfering substances and the complexity of the pesticide mixture. An effective co-solvent is used for efficient sample extraction, and TLC, column chromatography, and liquid-liquid partition methods are utilized for cleanup and preseparation of pesticides. Selective GC separations on two or more unlike columns and detection by electron capture, microcoulometric or electrolytic conductivity are employed in the final identification of the materials. A detailed discussion of sample handling, equipment, calculations, and reporting of results makes the method easily reproduced.

73-2272. Pattenden, G.; Crombie, L.; Hemesley, P. (Chem. Dept., The University, Nottingham NG7 2RD, England). **The mass spectra of the pyrethrins and related compounds.** *Organ. Mass Spectrom. 7(6): 719-735; 1973.* (7 references)

The use of a combination of GLC and mass spectrometry has been found the most unambiguous means of determining trace amounts of pyrethrin esters. In their mass spectra the six esters show weak molecular ions. The primary fragments observed in each spectrum are a result of fission at the ester linkage. The pyrethrin I's display a characteristic group of three fragment ions corresponding to $[RC_9H_9O]^+$, $[RC_9H_{10}O]^+$ and to $[RC_9H_{10}O]^+$ (R=CH_3, C_2H_5 and C_2H_3). Allethrin also displays these characteristic ions. A trio of fragment ions separated by one mass unit and corresponding to the ions observed in the spectra of the pyrethrin I esters dominate the mass spectra of the pyrethrin II esters. Main fragmentation paths seen in authentic *cis*-rethrones are reproduced in spectra of pyrethrin esters since these are the same as the fragments mentioned above. Considerable characterization of compounds related to pyrethrins has been accomplished in terms of mass spectral data.

73-2273. Qadri, S. S. H. (Dept. Zool., Post-Grad. Cent., Warangal, A. P. India). **Insects suitable for the microbioassay of insecticide residues.** *Pesticides 7(3): 22-23; 1973.*

The suitability of adults of *Musca domestica nebulo* Fabr., *Tribolium castaneum* Hbst., *Sitophilus oryzae* L., *Callosobruchus chinensis* L., and the larvae of *T. castaneum* Hbst. and *Bombyx mori* L. was investigated for microbioassay of lindane, malathion, Sumithion (fenitrothion), diazinon, and parathion. Susceptibility of age effect and order of susceptibility to lindane, malathion, and diazinon were observed with *T. castaneum*. The susceptibilities of larvae of first instar *T. castaneum* to diazinon; second instar *B. mori* to lindane, malathion, sumithion, diazinon, and parathion; and of 4-wk-old adults of *T. castaneum* and *S. oryzae* and 7-day-old adults of *B. chinensis* to diazinon, Sumithion, and malathion ranged from 2.0 to 0.047 μg and could be utilized in microbioassays if great accuracy was not required.

73-2274. Scudamore, K. A.; Heuser, S. G. (Min. of Agr., Fish. and Food, Pest Infestation Control Lab., London Rd., Slough, Bucks, England). **Determination of carbon tetrachloride in fumigated cereal grains during storage.** *Pestic. Sci. 4(1): 1-12; 1973.* (29 references)

Alternative methods for the extraction of unchanged carbon tetrachloride residues from fumigated whole and ground wheat and maize were examined and compared. Amounts of carbon tetrachloride extracted were determined by gas-liquid chromatography using two separate columns and an electron-capture detector. A portion of any carbon tetrachloride residue was converted to chloroform by a steam distillation extraction method but not when a cold solvent extraction process was used. In addition, the effectiveness of removal of carbon tetrachloride from wheat and maize during a 3.5 hr steam distillation was progressively lessened, in comparison with the cold extraction process, as the length of time that residual fumigant had been associated with the grain increased. The rate of elimination of carbon tetrachloride from wheat and maize during airing at two temperatures was determined, and though partially dependent on the temperature of fumigation, airing was consistently more rapid at 25°C than at 10°C. Initial residues of 200 to 400 parts/million in whole grains fell to 1 to 10 parts/million when aired for six months in thin layers at 25°C and to 5 to 50 parts/million at 10°C. Residues in wheat disappeared more rapidly than those in maize. Grinding initially caused a sharp reduction in carbon tetrachloride content, but subsequent airing rates were little faster than those of the whole grains. It is concluded that complete elimination of trace amounts of carbon tetrachloride from products of treated grain is unlikely even after milling, but the toxicological significance of such residues is uncertain. (Author abstract by permission)

73-2275. Edmunds, J. W.; Lee, D. F.; Nickels, M. L. (Min. of Agr., Fish. and Food, Plant Pathol., Hatching Green, Harpenden, Hertfordshire, England). **Determination of 2,3,7,8-tetrachlorodibenzo-1,4-dioxin in 2,4,5-trichlorophenoxyacetic acid and 2,4,5-T alkyl ester herbicides.** *Pestic. Sci. 4(1): 101-105; 1973.* (8 references)

A gas chromatographic method for the determination of 2,3,7,8-tetrachlorodibenzo-1,4-dioxin in 2,4,5-trichlorophenoxyacetic acid and 2,4,5-T alkyl ester herbicides is described. Of 80 samples of 100% 2,4,5-trichlorophenoxyacetic acid alkyl ester herbicides 25 contained less than 0.05 part/million and 75 less than 0.5 part/million 2,3,7,8-tetrachlorodibenzo-1,4-dioxin. Of 32 samples of 50% formulations of 2,4,5-T alkyl esters in mineral oil, 8 contained less than 0.05 part/million and 18 less than 0.5 part/million tetrachlorodioxin. The maximum found was 28.3 part/million. (Author abstract by permission)

73-2276. Muir, D. C. G.; Baker, B. E. (Dept. of Agr. Chem., McGill Univ., Montreal, Canada). **Pesticide residues in soil and foodstuff. I. Chlorinated pesticides in cattle and milk produced in orchard and non-orchard areas.** *Pestic. Sci. 4(1): 113-119; 1973.* (15 references)

Milk, hay, and silage produced in orchard and non-orchard areas were analyzed for organochlorine pesticide residues. Aliquots (150 ml) of milk samples were extracted with acetone-hexane, 1:1 volume. Two percent deactivated Florisil was used for clean-up and separation. The column was eluted with 300 ml hexane and then three different mixtures of dichloromethane and hexane, a technique which separated dieldrin from *p,p'*-DDE and heptachlor epoxide from *o,p'*-DDE or *p,p'*-TDE olefin. Hay and silage samples were ground and water added to 80%. Acetonitrile and hexane were used for extraction and partitioning which were followed by Florisil column clean up. Residues were determined using a gas chromatograph equipped with an electron capture detector. Evidence for the presence of a pesticide was given by its appearance in a particular eluate, the retention time on two different GLC columns, and TLC behavior. Dieldrin's presence was confirmed by reaction with HBr in acetic anhydride, those of *p,p'*-TDE and *p,p'*-DDT by dehydrochlorination, and those of lindane, hepatchlor epoxide, *p,p'*-DDE and *p,p'*-TDE olefin by TLC. Recoveries were high. Results indicated that levels were higher in milk (almost double) and hay (30% up to sevenfold) from orchard areas than from non-orchard areas. Silage residues scarcely differed. Milk collected after cattle had been grazing in an orchard for 3-4 days had 10% more *p,p'*-DDT and 2-,3-,7- and 15-fold more *p,p'*-DDE, *p,p'*-TDE and heptachlor epoxide, lindane, and *p,p'*-TDE olefin than did the samples collected one day before the cattle grazed in the orchard.

73-2277. Masud, Z.; Batora, V.; Kovacicova, J. (Pestic. Lab., Dept. of Plant Protect., Malir Halt, Karachi-27, Pakistan). **Gel filtration clean-up for multi-residues of organophosphorus pesticides in rice.** *Pestic. Sci. 4(1): 131-136; 1973.* (16 references)

Gel filtration on Bio-Beads S-X8 and Sephadex LH-20 columns has been sucessfully applied to the clean-up of multiple residues of malathion, fenitrothion, and dichlorvos in rice. Recoveries were checked by gas-liquid chromatography using CsBr-thermionic detector and ranged from 77 to 104% at 5 mg/kg level. The clean-up was found to be highly effective. No interference was observed in the gas chromatograms. (Author abstract by permission)

73-2278. Ong, B. Y.; Falk, R. H.; Bayer, D. E. (Dept. Botany, Univ. California, Davis, CA 95616). **Scanning electron microscope observations of herbicide dispersal using cathodoluminescence as the detection mode.** *Plant Physiol. 51(2): 415-420; 1973.* (10 references)

A rapid method which utilizes the fluorescence of a number of herbicides under electron bombardment was developed for spatially localizing herbicides on leaf surfaces. All 17 of the compounds tested produced some fluorescence, and naptalam, diuron, fenuron, dicamba, and MCPA were considered excellent in this respect. Increases in fluorescence of the benzene were observed when the subtituents were $-NH_2$, -NHR, NR_2, -COOH, -OH, $-OCH_3$, $-OC_2H_5$, and -CN. Using a scanning electron microscope with an EMI type 6255B CL photomultiplier tube, the fluorescence or cathodoluminescence was recorded for leaf surfaces treated with the herbicides. The method appears to have potential for measuring the persistence of herbicides in treated fields.

73-2279. Flattau, J.; Rutkowska, K. (Inst. for Ind. Chem. Warsaw, Poland). **Proba zastosowania detektora plomieniowo-jonizacyjnego do oceny skutecznosci dzialania filtropochlaniaczy wobec pestycydow. [Attempts of using a flame ionization detector for evaluating the efficiency of filter canisters for absorbing pesticides.]** *Pr. Cent. Inst. Ochr. Pr. 22(72): 41-48; 1972.* (5 references) (Polish)

The possibility of detecting traces of organophosphorus pesticides behind the filter canister by means of a flame ionization detector was investigated. Using a chromatogram obtained for the sample of pesticide concentrate, a simple method was developed for the evaluation of the sensitivity of a flame ionization detector with frontal supply of pesticide contaminated air. The results indicated that the flame ionization detector is not sensitive enough to detect the maximum admissible concentration of organophosphorus pesticides.

73-2280. Bonelli, E. J. (Finnigan Corp., Sunnyvale, CA). **GC/MS techniques for the determination of interferences in pesticide analysis.** *Proc. Inst. Environ. Sci.*

Annu. Tech. Meet. 18: 251-254; 1972. (6 references)

A GC/MS was used to analyze various mixtures of pesticides, polychlorinated biphenyls (PCBs), and chlorinated naphthalenes. The *p,p'*-DDE/*p,p'*-DDT/PCB mixture was chromatographed on a column packed with 3% OV-1 on 60/80 mesh Gas Chrom Q. The mixtures of organochlorine pesticides with Arochlor 1254 and Halowax 1014 were chromatographed on columns containing 5% OV-17 on 60/80 mesh Gas Chrom Q. For peak monitoring voltages corresponding to the mass ions of interest were applied to the quadrupole mass filter, allowing the monitoring of up to eight ion peaks over the entire mass range of the MS. Quantitation of levels in the nanogram and picogram range was achieved by comparing the area under one or more of the eight peaks to the area of a peak of a known amount of standard. The use of a computer facilitated interpretation of data.

73-2281. Machin, A. F. (Biochem. Dept., Min. Agr., Fish. Food, Cent. Vet. Lab., New Haw, Weybridge, Surrey, England). **Some procedures for isolating toxic metabolites of phosphorothionate pesticides from animal tissues and fluids.** *Proc. Soc. Anal. Chem. 10(4): 92-93; 1973.* (10 references)

Three metabolites of diazinon were separated using techniques including thin-layer, gas, column, and high-pressure liquid chromatography. The methods used should be applicable to metabolites of phosphorothionate pesticides. Thin layer chromatography has been used for isolating and determining toxic metabolites by their inhibition of esterases. It also provides information about the phosphoryl moiety. The two hydroxy derivatives of diazinon from urine were separated by repeated TLC. GC isolated microgram quantities of metabolites. Phosphorus-containing residues were detected with a sensitive thermionic detector. Although column chromatography is tedious, it is the only technique capable of separating dehydrodiazinon from larger amounts of diazinon and fat. The two hydroxydiazinons were successfully separated with high-pressure liquid chromatography.

73-2282. Stevenson, D. S. (Chem. Dept., Univ. Glasgow, W.2, Scotland). **Application of liquid-gel chromatography to the analytical characterization of pyrethrum extract.** *Pyrethrum Post 11(3): 90-93; 1972.* (9 references)

Two systems of liquid-gel chromatography were investigated for analysis of pyrethrum extracts. The first utilized 56% hydroxyalkoxypropyl Sephadex G-25. The material was eluted as one fraction, without resolution, into cinerin, jasmolin, and pyrethrin components, with a benzene-light petroleum solvent system. The method is convenient for determining total pyrethrin content, and individual components can be monitored by GC analysis of the pyrethrin fraction. Using this system β-amyrin and β-sitosterol were identified as major components of the sterol fraction in addition to taraxasterol. Partial resolution of pyrethrins was achieved using 64% hydroethoxypropyl Sephadex G-25 and an isopropanol-light petroleum solvent system. This separation is being investigated further.

See also

73-2032 **73-2067** **73-2075** **73-2081** **73-2119**

HAPAB QUARTERLY INDEX

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PREFACE

The basic design for this index was supplied by the Technical Services Division, EPA Office of Pesticide Programs. It consists of two subject indexes (compound and concept) and two author indexes (personal and corporate).

Compounds are indexed by their common names; where other compound designations are used by authors, these synonyms appear with the notation to *see* the common name. Articles referring, in general, to structural or functional chemical classes are indexed under these broader headings (e.g., organochlorines, herbicides, etc.).

Subheadings in the compound index are identical with the main headings in the concept index, the latter having a maximum of three levels of specificity in addition to the compound.

Following is a cross-referenced guide to the subject headings:

Alternative Controls
Analysis
Antidotes (*see* Treatment of Poisoning)
Associations/Organizations
Beneficial Effects
Biochemical Effects (*see also* Enzyme Activity)
Blood/Body Fluids
Carcinogenesis
Cardiovascular System
Cholinesterase (*see* Enzyme Activity)
Cholinesterase-Measurement (*see* Analysis - Enzyme-Cholinesterase)
Chromatography (*see* Analysis - Chromatography)
Conferences
Cytological Effects
Decontamination (*see* Residue Removal)
Degradation (*see* Residue Degradation *and* Metabolism)
Dermal Effects (*see* Integument)
Diet (*see* Factors Influencing Metabolism/Toxicity - Nutritional State)
Digestive System
Domestic Animals (*see* Toxicity/Domestic Animals *and* Residues/Domestic Animals)
Economics
Education/Training (*see also* Prevention)
Embryology (*see* Reproduction/Growth - Embryo/Fetus)
Endocrine System (*see also* Reproduction/Growth)
Environmental Pollution
Enzyme Activity (*see also* Biochemical Effects)
Excretory System
Experimental Animals (*see* Toxicity/Experimental Animals)
Experimental Design
Factors Influencing Metabolism/Toxicity
Food and Feed (*see* Residues/Food and Feed)
Human exposure (*see* Toxicity/Humans *and* Residues/Humans)
Immunology
Interactions (*see* Factors Influencing Metabolism/Toxicity)
Laws and Regulations (*see also* Prevention)
LD50 (*see* Toxicity/Experimental Animals)
Metabolism (*see also* Residue Degradation)
Microscopy, light and electron (*see* Cytology)
Morbidity and Mortality Statistics
Musculoskeletal System
Mutagenesis/Teratogenesis (*see also* Reproductive System)
Nervous System
New Journals
Nomenclature
Photodecomposition (*see also* Residue Degradation)
Plants (*see* Residues/Plants)
Prevention (*see also* Laws and Regulations *and* Education/Training)
Reproduction/Growth (*see also* Endocrine System *and* Mutagenesis/Teratogenesis)
Residue Degradation (*see also* Photodecomposition *and* Metabolism)
Residue Dynamics
Residue Removal
Residues/Air
Residues/Domestic Animals
Residues/Food and Feed
Residues/Human
Residues/Plants
Residues/Soil
Residues/Wildlife
Respiratory System
Reticuloendothelial System
Reviews
Sensory System
Skeleton (*see* Musculoskeletal System)
Soil (*see* Residues/Soil)
Teratogenesis (*see* Mutagenesis/Teratogenesis)
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73-1647
73-1899
73-1978
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73-1748
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73-1737
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73-2005
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73-2191
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73-1730
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73-1546
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73-1851
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73-1585
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73-2047
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73-1937
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73-1579
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73-1529
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73-2114
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73-1906
73-1949
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73-2048
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73-2024
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73-1925
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73-1947
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73-2086
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73-1942
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73-1541
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73-1527
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73-2079
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73-1641
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73-1716
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73-1686
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73-1791
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73-1617
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73-1685
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73-1831
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73-1751
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73-1641
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73-1578
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73-1831
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73-1812
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73-2112
73-2222
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73-1972
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73-1868
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73-1900
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73-1599
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73-1616
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73-2111
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73-1706
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73-2089
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73-1994
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73-1853
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73-1762
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73-1875
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73-1774
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73-1711
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73-1634
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73-2194
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73-1587
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73-1546
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73-1994
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73-2143
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73-2235
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73-2272
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73-1820
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73-1819
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73-2067
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73-1800
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73-1790
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73-1656

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73-1623
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73-1729
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73-1661
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73-1689
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73-1718
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73-2196
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73-1632
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73-1903
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73-1546
73-2130
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73-1886
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73-1927
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73-2224
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73-1694
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73-2203
73-2204
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73-1962
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73-1653
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73-1729
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73-1756
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73-1594
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73-2048
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73-2187
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73-1698
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73-1747
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73-1978
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73-1816
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73-1651
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73-1812
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73-1739
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73-1632
73-1680
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73-2260
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73-1811
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73-1883
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73-1849
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73-1822
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73-2136
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73-1742
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73-2014
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73-1824
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73-1602
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73-2058
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73-2048
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73-1776
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73-1629
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73-2087
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73-2019
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73-1645
73-1972
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73-1848
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73-2275
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73-1546
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73-2208
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73-1586
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73-2138
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73-2214
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73-1580
73-1581
73-1992
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73-1960
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73-1702
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73-1913
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73-1916
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73-1684
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73-1566
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73-1644
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73-2016
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73-2192
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73-1641
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73-2080
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73-1955
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73-1662
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73-2193
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73-1827
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73-1831
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73-2241
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73-1857
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73-1710
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73-2180
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73-1617
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73-2107
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73-2279
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73-1961
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73-1710
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73-1835
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73-1998
73-2239
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73-1895
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73-1702
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73-2261
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73-2255
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73-1750
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73-2248
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73-1937
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73-2072
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73-2252
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73-1806
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73-1527
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73-1631
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73-1637
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73-2018
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73-1962
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73-1936
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73-1626
73-1918
73-2187
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73-1946
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73-1876
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73-1859
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73-1582
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73-1560
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73-1527
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73-1703
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73-2227
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73-1710
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73-1740
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73-1526
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73-1538
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73-2028
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73-2216
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73-1828
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73-1766
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73-1674
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73-1923
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73-1891
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73-1671
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73-2146
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73-2087
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73-1984
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73-1712
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73-2174
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73-2018
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73-2092
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73-1667
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73-1644
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73-2098
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73-2072
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73-2138
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73-1926
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73-1927
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73-2230
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73-2191
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73-1853
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73-1704
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73-2254
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73-1818
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73-1818
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73-1784
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73-1582
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73-1985
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73-1691
73-1699
73-1952
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73-1874
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73-1741
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73-1575
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73-1627
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73-1996
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73-1577
73-2046
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73-2157
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73-1631
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73-1610
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73-1810
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73-1765
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73-1914
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73-2123
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73-1675
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73-1829
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73-1827
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73-1910
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73-1960
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73-2008
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73-1857
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73-1832
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73-1657
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73-1690
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73-1927
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73-1813
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73-2069
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73-2025
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73-1679
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73-1840
73-2103
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73-1637
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73-2016
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73-1888
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73-1973
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73-2245
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73-1590
73-2049
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73-2160
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73-1976
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73-1699
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73-1630
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73-1644
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73-2249
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73-2053
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73-1583
73-2113
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73-1878
73-1879
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73-1797
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73-1797
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73-1676
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73-1622
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73-1624
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73-2272
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73-1681
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73-1540
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73-1641
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73-2147
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73-1882
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73-2274
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73-1875
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73-1833
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73-2107
73-2108
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73-1710
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73-1936
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73-1643
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73-1552
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73-2011
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73-2188
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73-2013
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73-1898

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73-2001
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73-1988
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73-1649
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73-2071
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73-2245
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73-2106
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73-1965
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73-1871
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73-1693
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73-1939
73-2110
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73-1767
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73-1618
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73-1815
73-1846
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73-1982
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73-1948
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73-1940
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73-1909
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73-1776
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73-1610
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72-2168
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73-1618
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73-1866
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73-2160
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73-1799
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73-1780
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73-2267
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73-1872
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73-2051
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73-1720
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73-1685
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73-1924
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73-2188
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73-1822
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73-1756
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73-2219
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73-1773
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73-1850
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73-2189
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73-1856
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73-2206
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73-1709
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73-2266
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73-1819
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73-1958
73-2218
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73-1582
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73-1562
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73-2189
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73-1587
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73-1825
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73-2238
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73-1699
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73-1831
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73-1766
73-1823
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73-1999
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73-1597
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73-2180
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73-2138
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73-1957
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73-1805
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73-2006
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73-1554
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73-1705
73-2240
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73-2046
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73-1673
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73-2096
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73-1952
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73-1870
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73-1537
73-2097
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73-1915
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73-1612
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73-2258
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73-1682
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73-1611
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73-1723
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73-2007
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73-1610
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73-1800
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73-2054
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73-2076
73-2237
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73-2219
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73-2046
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73-1725
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73-1847
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73-1670
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73-1859
73-1926
73-2105
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73-1890
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73-1810
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73-1801
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73-1889
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73-1779
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73-2139
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73-1954
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73-2063
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73-1776
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73-2106
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73-2106
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73-2085
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73-1604
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73-1738
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73-1576
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73-2247
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73-2202
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73-1897
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73-2111
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73-2208
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73-1727
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73-1537
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72-2168
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73-2001
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73-1955
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73-2160
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73-2022
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73-2072
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73-1642
73-1643
73-1749
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73-1963
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73-1615
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73-1713
73-1738
73-1754
73-1755
73-2184
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73-1844
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73-2213
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73-1566
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73-1825

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73-1666
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73-2258
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73-1613
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73-1600
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73-1916
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73-1722
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73-1690
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73-2198
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73-1981
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73-2083
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73-1550
73-1642
73-1643
73-1749
73-2031
73-2077
73-2263
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73-1770
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73-2258
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73-2277
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73-1625
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73-2213
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73-1892
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73-1611
73-1808
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73-2015
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73-1896
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73-1626
73-2187
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73-1870
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73-1830
73-2090
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73-1697
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73-1569
73-2026
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73-1942
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73-2101
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73-1948
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73-1752
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73-2188
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73-1748
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73-2092
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73-2024
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73-1872
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73-2091
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73-1738
73-2184
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73-1734
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73-1737
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73-1611
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73-1772
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73-1996
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73-1983
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73-1764
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73-1698
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73-2153
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73-1728
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73-2252
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73-2123
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73-1707
73-1974
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73-2114
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73-2275
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73-2187
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73-2116
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73-1582
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73-1844
73-1846
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73-1815
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73-1825
73-2088
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73-1648
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73-1745
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73-2252
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73-1800
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73-1551
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73-1936
73-1949
73-2066
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73-1967
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73-1968
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73-2164
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73-2132
73-2135
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73-1616
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73-1710
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73-1830
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73-1880
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73-1855
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73-1714
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73-1716
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73-1620
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73-2105
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73-2055
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73-2264
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73-2027
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73-1674
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73-1894
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73-1821
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73-2106
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73-1729
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73-1905
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73-1708
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73-2033
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73-1770
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73-2262
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73-1891
73-1938
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73-1709
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73-1639
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73-1897
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73-1583
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73-1903
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73-2022
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73-1857
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73-1593
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73-1389
73-2291
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72-2168
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73-1954
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73-1857
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73-2118
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73-1731
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73-1917
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73-1527
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73-2163
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73-1578
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73-1829
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73-1638
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73-1976
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73-2012
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73-2203
73-2204
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73-2251
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73-1816
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73-2188
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73-1648

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73-1700
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73-1720
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73-1582
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73-2090
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73-1969
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73-2277
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73-1581
73-1992
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73-1718
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73-1719
73-2236
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73-1610
73-2148
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73-1609
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73-1523
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73-1881
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73-1589
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73-1980
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73-1620
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73-1800
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73-1922
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73-1928
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73-1756
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73-1693
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73-1792
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73-1747
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73-1763
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73-2009
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73-2265
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73-1778
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73-1841
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73-1998
73-2239
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73-1920
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73-2157
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73-1531
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73-2215
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73-1652
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73-2007
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73-1794
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73-1746
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73-2157
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73-1943
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73-1785
73-2217
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73-2196
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73-1894
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73-2103
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73-1789
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73-1888
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73-2065
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73-1715
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73-2185
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73-1594
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73-1635
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73-1606
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73-1577
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73-1609
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73-1717
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73-2104
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73-1948
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73-2120
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73-1644
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73-2177
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73-2232
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73-1640
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73-1547
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73-2072
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73-2037
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73-2101
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73-1546
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73-1803
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73-1886
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73-2232
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73-1601
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73-2212
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73-1852
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73-1557
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73-2139
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73-2137
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73-1735
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73-2173
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73-2268
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73-2011
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73-2244
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73-1923
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73-1929
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73-2179
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73-1720
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73-2268
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73-1869
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73-2016
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73-1591
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73-1887
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73-1862
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73-1683
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73-1960
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73-1857
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73-2092
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73-1621
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73-1527
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73-2093
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73-1857
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73-2133
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73-1947
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73-2275
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73-2211
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73-2205
73-2207
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73-1613
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73-1714
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73-1848
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73-2086
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73-1780
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73-1966
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73-1980
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73-1976
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73-1967
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73-2199
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73-1684
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73-1887

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73-1665
73-1943
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73-2184
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73-1836
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73-1745
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73-2154
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73-1861
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73-2171
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73-2220
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73-1744
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73-1809
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73-1882
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73-1872
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73-2278
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73-1964
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73-1605
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73-1702
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73-1610
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73-2120
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73-1901
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73-1597
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73-1902
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73-1935
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73-1965
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73-1781
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73-1826
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73-1917
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73-2262
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73-1843
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73-2195
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73-1704
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73-1578
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73-2272
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73-1605
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73-2001
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73-2242
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73-1707
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73-1740
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73-1927
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73-1710
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73-1693
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73-1622
73-1624
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73-1900
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73-1696
73-2245
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73-1863
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73-1924
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73-1567
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73-1931
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73-1904
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73-1626
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73-1725
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73-1630
73-2170
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73-1702
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73-1839
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73-1530
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73-2172
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73-1755
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73-1728
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73-1629
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73-2050
73-2065
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73-1613
73-1732
73-1983
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73-1675
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73-2095
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73-1709
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73-2017
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73-2243
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73-1971
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73-1594
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73-1878
73-1879
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73-1749
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73-1616
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73-2222
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73-1695
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73-2273
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73-1921
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73-1570
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73-1989
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73-2050
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73-1546
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73-1945
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73-1601
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73-2101
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73-2245
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73-1638
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73-1638
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73-2116
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73-1871
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73-1986
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73-1973
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73-1999
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73-1606
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73-2136
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73-1553
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73-1676
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73-1605
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73-1687
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73-2081
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73-2132
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73-1636
73-1660
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73-1788
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73-1935
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73-1568
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73-2055
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73-1768
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73-1527
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73-2032
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73-1997
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73-1550
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73-1539
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73-1777
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73-1930
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73-1569
73-2026
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73-1541
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73-1541
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73-2158
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73-2227
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73-1820
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73-2212

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73-1689
73-1944
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73-1541
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73-2279
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73-2228
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73-1940
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73-1623
73-2120
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73-2258
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73-2070
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73-1606
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73-1775
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73-1922
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73-1859
73-1926
73-2105
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73-1832
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73-1591
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73-2057
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73-1592
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73-1873
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73-1828
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73-1718
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73-1932
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73-1768
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73-1718
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73-2055
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73-1703
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73-1606
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73-2155
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73-1890
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73-1623
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73-1571
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73-1692
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73-2085
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73-1911
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73-1622
73-1624
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73-2230
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73-1633
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73-1864
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73-2150
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73-1565
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73-1749
73-2002
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73-1582
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73-1555
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73-2274
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73-1605
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73-2264
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73-2004
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73-2120
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73-1622
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73-2004
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73-1694
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73-1746
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73-1941
73-2231
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73-1601
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73-1717
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73-1993
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73-2169
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73-2238
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73-1860
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73-1542
73-1588
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73-2243
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73-1977
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73-2021
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73-1844
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73-1849
73-2123
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73-1776
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73-1888
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73-1873
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73-1926
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73-1757
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73-1614
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73-1740
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73-1739
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73-1572
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73-2190
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73-1984
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73-1916
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73-1801
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73-2243
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73-1966
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73-2220
73-2221
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73-1646
73-1691
73-1952
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73-2075
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73-1636
73-1934
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73-1663
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73-1860
73-2142
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73-2094
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73-1619
73-1979
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73-1693
73-1705
73-2240
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73-2244
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73-2167
73-2172
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73-1933
73-1934
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73-1893
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73-2105
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73-1837
73-2093
73-2094
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73-1959
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73-2023
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73-2178
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73-1572
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73-1542
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73-2054
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73-1942
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73-2114
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73-1688
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73-1650
73-1912
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73-1843
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73-1945
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73-1884
73-1885
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73-1858
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73-2132
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73-2132
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73-1608
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73-1959
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73-1567
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73-1928
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73-1896
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73-1726
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73-1659

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73-2176
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73-1978
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73-2200
73-2210
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73-1583
73-2113
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73-1767
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73-1542
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73-1668
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73-2162
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73-1580
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73-1802
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73-1590
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73-1645
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73-1807
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73-1664
73-1704
73-1920
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73-1724
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73-2282
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73-1995
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73-1573
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73-1920
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73-1801
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73-1759
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73-1936
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73-2145
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73-1700
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73-2183
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73-2223
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73-1701
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73-1947
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73-2265
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73-1810
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73-1736
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73-2202
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73-1868
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73-2069
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73-1723
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73-1556
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73-2121
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73-1836
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73-1556
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73-1646
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73-2200
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73-2005
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73-1568
73-2056
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73-2160
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73-2029
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73-2104
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73-1614
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73-2269
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73-1610
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73-1744
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73-1687
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73-1776
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73-1536
73-2120
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73-2159
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73-1598
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73-1564
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73-1591
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73-2086
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73-1797
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73-1590
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73-1833
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73-2111
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73-2209
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73-2025
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73-1695
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73-1620
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73-1769
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73-1840
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73-2107
73-2108
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73-1567
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73-1724
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73-2256
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73-1594
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73-1747
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73-1708
73-1886
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73-1749
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73-1720
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73-1984
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73-1706
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73-1798
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73-1720
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73-1721
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73-2084
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73-1840
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73-1594
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73-1945
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73-1796
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73-1873
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73-1970
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73-1568
73-2056
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73-2073
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73-1572
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73-2086
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73-1750
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73-1677
73-1678
73-2229
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73-1525
73-1528
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73-1773
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73-1678
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73-2206
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73-1677
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73-1901
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73-2030
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73-2087
73-2089
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73-1669
73-1933
73-1934
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73-1625
73-1892
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73-2152
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73-1887
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73-1756
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73-1977
Verblyudova, N. I.
73-1754
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73-1688
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73-1672
Versteeg, J. P. J.
73-1850
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73-1774
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73-1699
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73-1962
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73-1870
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73-1630
73-2170
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73-2138

Vinckx, E.
73-1731
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73-1772
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73-1692
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73-1638
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73-1892
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73-1774
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73-2198
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73-1775
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73-1715
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73-1741
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73-1818
73-1842
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73-1591
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73-1989
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